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Jaclynn Elkind
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A Cross-Sectional fMRI Study Of Risk Factors Of Obesity In Emerging Adult Women And The Moderating Role Of Childhood Maltreatment

Abstract

Background: Obesity is a prevalent public health issue and affects more than 40 percent of adults in the United States. Women are more likely to be classified as obese than men, and are more likely to report disordered eating behaviors, particularly in young adulthood. Early life stressors, such as child maltreatment, can impact brain development in the areas related to decision-making and reward-driven behaviors like eating, which may contribute to the growing obesity epidemic. Neurobiological studies have shown that individuals who exhibit greater neural reactivity to highly palatable, rewarding foods who have poor impulse control may be more likely to engage in hedonic eating. This may be more pronounced in people with experiences of childhood maltreatment.

Methods: This dissertation will: 1) systematically review the relationship between childhood maltreatment and disordered eating in emerging adult women; 2) assess the relationship between hedonic hunger and neural responses to food cues; and 3) evaluate the relationship between impulsivity and neural responses to food cues. A moderation analysis will explore if the relationship between the neural response to food cues and hedonic hunger/impulsivity are strengthened by experiences of childhood maltreatment. A secondary data analysis was performed on a sample of emerging adult (18-24 years) women who participated in a two-day study using survey measures and functional magnetic resonance imaging to evaluate hedonic hunger, impulsivity and experiences of childhood maltreatment, and to measure how the brain responds to passive observation of visual food cues.

Results: Analysis of the literature revealed an association between experiences of childhood maltreatment and disordered eating behaviors in emerging adult women. Analysis of the fMRI data demonstrated differences in neural response to novel and repeated cues was associated with hedonic hunger, but not impulsivity in areas associated with executive control and interoceptive awareness. Experiences of childhood maltreatment did not alter these relationships.

Conclusion: The results from this study expand upon the current understanding of neurobiological vulnerabilities associated with risk for obesity in emerging adult women. Specifically, differences in neural response to novel and repeated cues may contribute to hedonic-driven eating behavior. These results require further exploration but will help to inform targeted screening and the development of tailored interventions to help those most at risk for obesity.

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A CROSS-SECTIONAL FMRI STUDY OF RISK FACTORS OF OBESITY IN
EMERGING ADULT WOMEN AND THE MODERATING ROLE OF CHILDHOOD
MALTREATMENT

Jaclynn Elkind

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A CROSS-SECTIONAL FMRI STUDY OF RISK FACTORS OF OBESITY IN
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MALTREATMENT

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Jaclynn Elkind, MSN, RN

DEDICATION

To Rob and our kids, and my family and friends, without whom this would not have been possible.

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ABSTRACT

A CROSS-SECTIONAL FMRI STUDY OF RISK FACTORS OF OBESITY IN EMERGING ADULT WOMEN AND THE MODERATING ROLE OF CHILDHOOD MALTREATMENT

Jaclynn Elkind

Anne Teitelman

Background: Obesity is a prevalent public health issue and affects more than 40 percent of adults in the United States. Women are more likely to be classified as obese than men, and are more likely to report disordered eating behaviors, particularly in young adulthood. Early life stressors, such as child maltreatment, can impact brain development in the areas related to decision-making and reward-driven behaviors like eating, which may contribute to the growing obesity epidemic. Neurobiological studies have shown that individuals who exhibit greater neural reactivity to highly palatable, rewarding foods who have poor impulse control may be more likely to engage in hedonic eating. This may be more pronounced in people with experiences of childhood maltreatment.

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Conclusion: The results from this study expand upon the current understanding of neurobiological vulnerabilities associated with risk for obesity in emerging adult women. Specifically, differences in neural response to novel and repeated cues may contribute to hedonic-driven eating behavior. These results require further exploration but will help to inform targeted screening and the development of tailored interventions to help those most at risk for obesity.

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Chapter 1

Introduction

Obesity, classified as a body mass index (BMI) of 30 kg/m² or greater, is a prevalent public health issue that results in 2.8 million premature deaths worldwide each year (World Health Organization, 2018). In the United States, obesity costs approximately \$149.4 billion dollars per year (Kim & Basu, 2016) and reduces life expectancy by an estimated 5 to 20 years (Fontaine et al., 2003). Obesity affects approximately 42.4% of adults and women are more likely to have obesity than men (40.4% women versus 35% men (Hales et al., 2017)). Further, women with obesity also report higher levels of disordered eating behaviors (Darby et al., 2007; Striegel-Moore et al., 2009) and are more likely than men to be diagnosed with an eating disorder (Hudson et al., 2007). This complicates the ability to provide adequate intervention and could therefore explain why the most common weight-loss interventions often fail in the long-term (Turk et al., 2009). As a result, studies of obesity and eating behaviors have shifted to explore the neurobiological implications in the development and maintenance of obesity.

Consideration of the neurobiological implications of any disease, including obesity, would be remiss to consider the impact of social determinants of health. Broadly, social determinants of health encompass the social structures and economic systems that contribute to health inequity. Although obesity rates are high in the general United States' population, disparities exist among racial/ethnic minorities and among economically disadvantaged people (Ogden et al., 2017). People from socioeconomically disadvantaged backgrounds are also at greater risk for adverse childhood experiences like child abuse which can alter brain development and function across the lifespan (Lawson

et al., 2017). These experiences are associated with physical and mental health impairment like depression and anxiety (Zielinski, 2009). Together, these may increase the likelihood of obesity (Herzog & Schmahl, 2018). Therefore, the purpose of this dissertation research is to explore the relationship between the neurobiological implications of eating behaviors and the impact of experiences of childhood maltreatment in emerging adult women, aged 18-24. Data were collected through an integrative review of the literature exploring the relationship between childhood maltreatment and disordered eating behaviors (**Chapter 2**) and a secondary analysis of data collected from the Brain and Urban Female Risk Study (BUFR 2) that explored how the neural response to food cues relates to “bottom up,” reward driven processes (mesolimbic) and “top down,” inhibitory related processes (mesocortical)- in emerging adult women. This secondary analysis assessed the correlation between the neural response to food cues and the Power of Food Scale scores as a measure of hedonic hunger (**Chapter 3**), and the Barrett Impulsivity Scale Version 10 (BIS-10) and the Affect-Congruent Go/NoGo task as measures of impulsivity and response inhibition (**Chapter 4**).

The **overall objectives** of this dissertation research were to: 1) explore disordered eating behaviors and its relationship with childhood maltreatment 2) examine the association between neural response to food cues and vulnerabilities associated with obesity (hedonic hunger, impulsivity and inhibition) in emerging adult women and 3) examine how experiences of childhood maltreatment inform the relationship between neural response to food cues and vulnerabilities for obesity. The **central hypothesis** of this dissertation research is that emerging adult women who have experienced childhood maltreatment will demonstrate increased vulnerability to food cues and risk factors

associated with obesity, as compared to women without experiences of child maltreatment. The specific aims were to:

Specific Aim 1: Complete an integrative review of publications in peer-reviewed journals evaluating the relationship between disordered eating behaviors and experiences of childhood maltreatment in emerging adult women. This review served as the foundation of the dissertation aims and hypothesis by providing an increased understanding of current trends in disordered eating behaviors and symptoms and the relationship to experiences of childhood maltreatment (**Chapter 2**).

Specific Aim 2: Examine the relationship between the neural response to images of palatable food cues and hedonic hunger in emerging adult women (**Chapter 3**).

Hypothesis 1: Higher hedonic hunger indicated by higher scores of the Power of Food Scale will correlate with increased activation in response to food cues in brain areas associated with reward and motivation.

Hypothesis 2: The relationship between hedonic hunger and the neural response to food cues in reward-associated areas will be moderated by childhood maltreatment, such that greater childhood maltreatment will be reflected in a stronger correlation between hedonic hunger and brain response to food cues.

Specific Aim 3: Examine the relationship between the neural response to images of palatable food cues and impulsivity and response inhibition in emerging adult women (**Chapter 4**).

Hypothesis 1: Increased activation in reward-areas of the brain will be associated with greater impulsivity (as measured by the Barratt Impulsivity Scale version

10), and decreased response inhibition (i.e. greater number of errors of commission on the Affect-Congruent Go/NoGo task).

Hypothesis 2: The relationship between impulsivity and response inhibition and neural response to food cues will be moderated by childhood maltreatment. The relationship between impulsivity, response inhibition, and neural response to food cues will be reflected in a stronger correlation between impulsivity, response inhibition, and the neural response to food cues.

With respect to **outcomes**, this dissertation fills a gap in the literature related to the relationship between vulnerabilities to overeating and the brain's response to food cues in emerging adult women from diverse sociodemographic backgrounds with and without experiences of childhood maltreatment. Identifying the neurobiological contributions to food-seeking behaviors may contribute to novel approaches of identifying people at greatest risk for obesity and interventions to prevent and/or treat obesity.

The remainder of this chapter will outline the theoretical framework, major concepts utilized in this dissertation, and the specific aims and rationale for each chapter.

Background

Theoretical Framework

The overarching framework of this dissertation research applies a life course perspective and draws from evidence that adverse childhood experiences negatively impact brain development and long-term health and wellness (Felitti et al., 1998; Shonkoff et al., 2012). Specific to the study of the brain, *Dual Process Models* (for a review McClure & Bickel, 2014; others) discuss the balance between “bottom up” motivational processes/impulses toward reward, in contrast to “top down” inhibitory abilities that resist rewarding stimuli to maintain long-term goals. When one, or both of

these two systems are not functioning adequately, behavior may not align with one's desired goals. Experiences of adverse childhood experiences, like childhood maltreatment, that are not adequately mitigated are one example of a disruption to early brain development and should be considered when examining eating behaviors in emerging adult women (**Figure 1**).

Emerging Adulthood

Emerging adulthood is characterized as the ages of 18 to 29 years and is a period of social and physiological growth and development (Arnett, Žukauskienė, & Sugimura, 2014). Socially, emerging adults typically demonstrate identity exploration and increased independence to make choices specifically about eating habits, exercise, and develop attitudes about their bodies that may be different from their parents' attitudes (Arnett et al., 2014; Nelson et al., 2008); however, this is also a period known for instability in housing and finances, preventing complete independence from parents or guardians and often contributing to increased stress. Physiologically, the emerging adult brain continues to undergo significant growth and development in the subcortical-reward based regions, associated with impulsivity, as well as growth in cortical regions, responsible for executive functions like attention, inhibition of impulses, working memory, and planning (Blakemore, 2012). Because of the continued development in these two areas, this time period is characterized by risky behaviors that threaten health and security (Sussman & Arnett, 2014). To mitigate the negative health effects, this may be a key time for intervention due to increased independence and freedom to make choices about one's health.

Early Childhood Adversity

There is evidence to suggest that adverse childhood experiences like childhood maltreatment, which includes harsh physical punishment, physical abuse, sexual abuse, emotional abuse, neglect, and exposure to intimate partner violence (IPV) until the age of 18 (Leeb et al. 2008), can result in a specific type of stress called toxic stress. Experiences of stress induce a physiological response involving activation of the brain's hypothalamic-pituitary-adrenocortical axis, which results in the release of hormones like cortisol. Release of these hormones is physiologically protective against stress, but prolonged, frequent, or strong activation of the body's physiological stress response results in toxic stress. Experiences of persistent elevated levels of cortisol from toxic stress during childhood are detrimental and can profoundly impact brain development in the limbic and cortical regions of the brain associated with learning and memory, emotions, and executive function (Fox et al., 2010). Impairment in these areas is associated with a variety of psychological and physical health outcomes, including eating disorders and obesity (Oh et al., 2018). Therefore, in order to develop tailored treatment and prevention strategies for disordered eating and obesity, it is critical to explore the brain mechanisms by which experiences of childhood maltreatment impact obesity-related vulnerabilities.

Disordered Eating Behaviors

The prevalence of disordered eating behaviors like binge eating, purging, dieting, and unhealthy weight loss practices that do not meet diagnostic thresholds are more prevalent than clinical eating disorders (Stephen et al., 2014). Disordered eating behaviors are associated with increased risk for clinically relevant eating disorders (Jacobi et al., 2004; Joiner Jr et al., 1997) and weight gain and/or obesity (Field et al., 2007; Neumark-Sztainer et al., 2006; Stice et al., 1999). Evidence shows that people that are classified as

overweight or obese are more likely to report disordered eating behaviors than people that are underweight or normal weight (Nagata et al., 2018) and these behaviors are especially prevalent in emerging adult women. Estimates show 20%-61% of females report disordered eating compared to 31%-39% for males (Croll et al., 2002; Delinsky & Wilson, 2008; Mintz & Betz, 1988; Striegel-Moore et al., 2009).

The independence that accompanies emerging adulthood gives individuals the freedom to make choices regarding eating habits, exercise, and attitudes about their bodies (Arnett et al., 2014; Nelson, Story, Larson, Neumark-Sztainer, & Lytle, 2008). Evidence suggests that emerging adults have lower levels of body satisfaction and higher levels of disordered eating behaviors (Delinsky & Wilson, 2008; Gillen & Lefkowitz, 2006). Although eating disorders typically begin in adolescents, there is evidence from longitudinal studies that these behaviors not only increase during emerging adulthood (Delinsky & Wilson, 2008), but adolescents will continue to engage in these behaviors beyond a 10-year-period (Goldschmidt et al., 2016; Pearson et al., 2017), suggesting that the emerging adult period may be a key time for identification and treatment.

A key finding from the clinical eating disorder literature is the increased prevalence of experiences of maltreatment in childhood. Women with clinical eating disorders are more likely to report experiences of maltreatment in childhood than women without eating disorders (Brewerton, 2007; Carlos M Grilo et al., 2005; Pignatelli et al., 2017; Rayworth et al., 2004), but it is unclear how experiences of maltreatment contribute to subclinical, disordered eating behaviors. Because disordered eating behaviors are more common than clinical eating disorders, it is important to understand the relationship between disordered eating behaviors and experiences of maltreatment in childhood.

Regulation of Food Intake

The brain plays a critical role in the integration of hormonal and neural signals that regulate energy homeostasis or energy balance to support the metabolic needs of the body (Volkow, Wang, Tomasi, & Baler, 2013; Woods, Seeley, Porte, & Schwartz, 1998). Specifically, there are two distinct, but overlapping neural pathways that are involved in the behavior of food intake, homeostatic regulation and hedonic regulation. **Homeostatic regulation** involves the maintenance of energy balance, which is the combination of energy expenditure and energy intake (Rossi & Stuber, 2018). In response to an energy deficit, peripheral hormones like insulin, leptin, and ghrelin signal to the hypothalamus that there is an energy deficit causing a feeling of hunger and food-seeking behavior (Woods et al., 1998). **Hedonic regulation** involves pleasure-based food consumption via activation of the brain's mesolimbic dopamine system (Lutter & Nestler, 2009), that includes the ventral tegmental area (VTA), nucleus accumbens (NAc), amygdala, and hippocampus (Russo & Nestler, 2013). This system can be activated by foods that are highly palatable (i.e. high in fat and sugar), emotional factors like stress or depression, or environmental factors such as cues in the environment or the availability of food (Patel & Schlundt, 2001). Activation of this system occurs via release of dopamine to produce a positive affect and increased "desire for food" and "hunger" (Wang et al., 2004). This desire and hunger is associated with increased food intake, even in the absence of homeostatic requirements (Kelley et al., 2002; Lowe & Butryn, 2007). Because true energy deficiency is rare in today's society, most of food consumption is today's society is the result of *hedonic hunger* (Lowe & Butryn, 2007). Therefore, one of the goals of this dissertation is to quantify the brain's mesolimbic activity in response to the passive

viewing of highly rewarding food cues using functional magnetic resonance imaging (fMRI), a noninvasive measure of brain activity that detects changes in blood oxygenation and flow that occurs in response to neural activity.

Food Cues

The food environment in the United States consists primarily of highly palatable, high-calorie foods that act as stimuli or cues to trigger food motivation and food-seeking. From the perspective of Pavlovian (classical) conditioning, food cues (sights, sounds, or smells) that reliably signal food reward can come to trigger physiologic events (e.g. dopamine release/activation of reward circuitry) that initially occur to the food reward itself (Berridge & Robinson, 1998; Schultz, 2006, 2010); these brain events may underlie desire, craving, and motivation to action -- e.g., food seeking (Wang et al., 2004). In most real-world situations, food-seeking is also often followed by food reward, further strengthening the behavioral responses into learned ‘habits’ by Instrumental (or operant) reinforcement (Everitt & Robbins, 2005; Volkow et al., 2013). Though both Pavlovian and Instrumental learning processes can shape food-seeking, this dissertation research uses a Pavlovian framework to explore individual differences in the brain response to food cue ‘triggers’. As there are substantial individual differences in the motivation to seek reward (Berridge & Robinson, 1998; Childress et al., 1993; Epstein, Temple, Roemmich, & Bouton, 2009; Schultz, 2010), identifying the underlying brain differences may help us identify (and eventually, treat) individuals who are vulnerable to cue-triggered over-eating.

Hedonic Hunger

Hedonic hunger is defined as the drive or motivation to seek food for the pleasure experienced from food consumption (Lowe & Butryn, 2007). Hedonic hunger is

hypothesized to be partly responsible for the rise in and the maintenance of obesity because of the availability of highly palatable foods and the reward associated with consumption of these foods (Brownell & Horgen, 2003; Lowe & Butryn, 2008). The Power of Food Scale (PFS) was developed as an objective measure of hedonic hunger or the motivation to seek out palatable food (Lowe & Butryn, 2007). It is used to assess the self-reported sensitivity to the rewarding properties of highly palatable food in the environment, not the consumption of food (Lowe & Butryn, 2007) and is associated with frequency and intensity of food craving (Forman et al., 2007), and with severity of binge eating in people with obesity (Ochner et al., 2009).

Highly palatable, energy dense foods are associated with overeating due to the reward or pleasure derived from consumption of this type of food, which, similar to drugs of abuse, activates the reward centers in the brain and promotes reward-driven or hedonic eating (Davis & Carter, 2009). Because of the brain's implication in reward-motivated behaviors like eating, imaging techniques may be used to study the brain response to environmental cues. Evidence from studies using fMRI and food cues suggests overeating in people with obesity compared to normal weight individuals differentially activates the reward-related, mesolimbic regions of the brain including striatum, midbrain, and orbitofrontal cortex (Kringelbach et al., 2003, Small et al., 2001, Stice, Burger, & Yokum, 2013). Notably, higher PFS scores are associated with heightened response to food cues in brain areas associated with somatosensory processing including the post-central gyrus (Burger et al., 2016), the right middle frontal gyrus and right parietal lobule (Jensen et al., 2016), and the insula (O'Doherty et al., 2001), suggesting that high hedonic hunger is related to the sensory properties of food cues. However, the

literature in this field primarily focused on white or Caucasian college students or people seeking treatment for obesity. Thus, the generalizability of these results does not extend to other individuals who are vulnerable to developing obesity, such as *emerging adult women not attending college, minority women, or those with a history childhood maltreatment*.

Inhibitory Control

Working with the mesolimbic dopamine system, the mesocortical pathway projects from the ventral tegmental area in the basal ganglia to the prefrontal cortex (Volkow et al., 2013). In addition to executive function tasks that involve reasoning, planning, and problem solving, this region is responsible for inhibitory control of behavior or dampening the motivation for appetitive, rewarding stimuli (Appelhans, 2009; Carr et al., 2011). Because behavior is tightly controlled by the brain, any disruptions in function can further increase susceptibility to rewarding stimuli and result in unwanted or unhealthy behaviors like hedonic (over-)eating. Evidence suggests that the inhibitory regions like the prefrontal cortex are particularly vulnerable to experiences of maltreatment in childhood (Hart & Rubia, 2012), resulting in altered structure and function (Hanson et al., 2013). Furthermore, evidence suggests that palatable food can override these inhibitory signals (Kaye et al., 2009). People with experiences of childhood maltreatment, who are more likely to have a compromised inhibitory system, may be even more vulnerable to rewarding stimuli (Teicher et al., 2016).

Deficient inhibitory processes may present additional vulnerabilities for people faced with an obesogenic environment (Hofmann et al., 2009; Metcalfe & Mischel, 1999). Inhibition can be measured using tasks like the “Go/NoGo” task that involves pressing a

button in response to frequent “Go” stimuli and refraining from pressing the button in response to infrequent “NoGo” or stop stimuli (Horn et al., 2003; Goldman et al., 2015, Table 1.2). Pressing the button to a NoGo stimulus reflects a failure of inhibition, which is a proxy measure for impulsivity, a multidimensional personality trait that is defined as a predisposition toward rapid, unplanned actions without regard for the possible negative consequences of the actions (Moeller et al., 2001).

Impulsive behavior is associated with rapid decision-making, inattention, lack of perseverance, acting without thinking, lack of planning, sensation seeking, and risk-taking (Moeller et al., 2001) and correlates with deficits in control over food intake (Dawe & Loxton, 2004; Schag et al., 2013; Waxman, 2009). Furthermore, lower response inhibition and self-report measures of impulsivity have been associated with increased calorie consumption (Guerrieri, Nederkoorn, Stankiewicz, et al., 2007), overweight, and obesity (Brooks et al., 2013; Nederkoorn, Braet, et al., 2006), but no studies have examined this relationship in emerging adult women, specifically, or considered the effect of experiences of childhood maltreatment.

Although there is evidence to suggest that impulsivity is associated with overeating and obesity, there is very limited evidence on the neural response to food cues and impulsivity and response inhibition in a community sample of emerging adult women. In addition, literature that specifically examines these associations in women with and without experiences of maltreatment is sparse. This dissertation research will be one of the first papers to specifically evaluate the relationship between neural response to food cues and impulsivity and response inhibition using the Affective Go/NoGo task in emerging adult women with the moderating effect of childhood maltreatment.

Study Design

This study will first explore the relationship between disordered eating behavior and symptoms and experiences of maltreatment in emerging adult women through an integrative review of the literature (Chapter 2). Subsequent analysis examines data collected from the Brain and Urban Female Risk Study (BUFR 2) to explore how the neural response to food cues relates to “bottom up” (mesolimbic) and “top down” (mesocortical)- related measures in emerging adult women. Analysis for Chapter 3 and Chapter 4 will utilize data from the Behavior in Urban Female Risk Study (BUFR 2). This study was designed to examine the neural response to neutral and evocative cues (food, sex, aversive) in emerging adult women (18-24) with varying degrees of risk for sexually transmitted infections (STIs)/HIV infection. Participants (n= 60) were recruited from a federally supported Title X (serving low income individuals) family planning clinic and from a nearby university, both of which were located in a large urban area in the northeast United States. Subjects participated in two sessions, typically scheduled on consecutive days. In the first session, participants completed informed consent, surveys, and demographic health information via pen and paper. For sensitive topics like intimate partner violence and adverse childhood experiences, Audio Computer Assisted Self-Interviewing (ACASI) was utilized for data collection in order to increase the accuracy of self-report data (Morrison-Beedy et al., 2006; Newman et al., 2002). In the second session, a functional magnetic resonance imaging (fMRI) scan was completed while participants performed selected tasks on a computer. Due to the nature of fMRI, participants were excluded if they had a history of a seizure disorder, were greater than

approximately 300 pounds, or were pregnant. Following completion of the study, participants were compensated 105 dollars.

The *overall objectives* of the proposed study are to examine the eating behaviors of emerging adult women and to explore the relationship between the brain response to food cues and vulnerabilities that may contribute to overeating. A secondary objective is to determine if the relationship between the brain response to food cues and vulnerabilities to overeating is altered by experiences of childhood maltreatment. This will be accomplished by 1.) examining the relationship between disordered eating behaviors in emerging adult women and experiences of maltreatment in childhood, 2.) examining neural response to food images compared to neutral using functional magnetic resonance imaging (fMRI) to determine if there is a relationship with hedonic hunger; and 3.) examining the neural response to food images compared to neutral using fMRI to determine if there is a relationship between response inhibition and impulsivity; and 4.) determine if either of these relationships from 2) and 3) is moderated by childhood maltreatment. The **central hypothesis** of this dissertation research is that emerging adult women that have experienced childhood maltreatment will demonstrate increased vulnerability to food cues and risk factors associated with overweight and obesity, as compared to women without experiences of child maltreatment.

Significance

Obesity is prominent public health problem for which there are many independent and interacting risk factors, causes, and few long-term treatments that are effective (Rossner et al., 2008; Wing & Hill, 2001). Although evidence shows increased impulsivity, lower inhibition, and hedonic eating are associated with overweight and

obesity (Hruby & Hu, 2015) it is unclear how experiences of childhood maltreatment may contribute to this relationship (Morris et al., 2015). Further, evidence demonstrates income and education, markers of socioeconomic status, are inversely associated with obesity risk in women (Brooks, Cedernaes, & Schioth, 2013; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006) and childhood maltreatment (Herrenkohl & Herrenkohl, 2007; Zielinski, 2009), but few studies examine how these interact from a neurobiological perspective. Therefore, this study will begin to explore these relationships.

An ability to identify specific areas of the brain associated with behavior will aid in identification and the development of targeted interventions (behavioral or pharmacologic). Nurses are in a unique position to drive these evidence based solutions because of their position in the community and schools, as nurse practitioners in the clinic, and as scientists and policymakers in healthcare and government. By understanding the interaction and contribution of neurobiology and social determinants of health, nurses can address obesity from a holistic approach and develop more effective strategies to reduce the burden of obesity and promote healthy eating behaviors.

Innovation

This dissertation takes a unique approach to the identification of biomarkers contributing to behaviors and associated risks for obesity. Utilization of fMRI to identify the neural correlates of behavior is an understudied field. This research will add to the literature examining neural vulnerabilities to overeating by exploring these associations in a diverse sample of young adult women. Furthermore, the neurobiological implications of childhood maltreatment and risk factors of obesity are limited. This study will explore

mechanisms that contribute to obesity with the goal of enhancing our ability to identify and treat outcomes with tailored and precise interventions.

Chapter Aims and Rationale

The major goals of this dissertation research were to: 1) explore disordered eating behaviors and symptoms that are associated with childhood maltreatment (**Chapter 2**); 2) examine the relationship between the neural response to food cues and vulnerabilities associated with obesity in emerging adult women (**Chapter 3**) and 3) examine how this relationship may be changed by experiences of childhood maltreatment (**Chapter 4**).

Chapter 2

Aim: Explore disordered eating behaviors and symptoms associated with childhood maltreatment.

Rationale: This review served as the foundation of the dissertation aims and hypothesis by providing an increased understanding of current trends in disordered eating behaviors and symptoms, and the relationship to experiences of childhood maltreatment. Disordered eating behaviors are more common than clinically-diagnosed eating disorders and are associated with experiences of childhood maltreatment. Although there is a great deal of research exploring the relationship between childhood maltreatment and clinical eating disorders, there is less research exploring non-clinical disordered eating behaviors and experiences childhood maltreatment. This review will be the first to review the literature that explores the relationship between disordered eating behaviors and experiences of childhood maltreatment.

Chapter 3

Aim: Examine the relationship between hedonic hunger and the brains response to palatable food cues in emerging adult women.

Rationale: Sensitivity to food cues as measured by increased activation in the reward regions of the brain may be associated with eating in the absence of hunger. Emerging adult women who report greater levels of hedonic hunger may be more likely to overeat, especially when presented with rewarding stimuli like images of food.

Chapter 4

Aim: Determine the relationship between impulsivity and response inhibition and the neural response to visual food cues.

Rationale: The prefrontal cortex is an area known to modulate impulsive decisions like eating in the absence of hunger or overeating. People with greater impulsivity and poorer inhibition will have decreased neural activation in areas brain associated with inhibition. These deficits are likely to be associated with greater impulsive behaviors. This paper will fill the gap in the literature by exploring the association between the neural response to food cues and impulsivity and response inhibition in a diverse cohort of emerging adult women. This paper will also explore whether experiences of maltreatment in childhood strengthen that relationship.

Figures and Tables

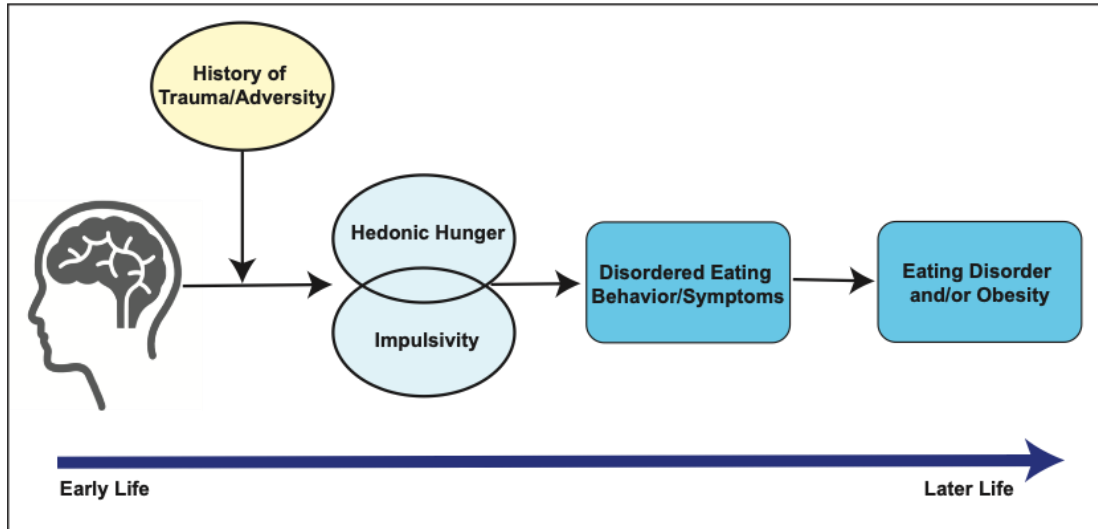


Figure 1.1 Theoretical Model

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Chapter 2

The Relationship Between Disordered Eating and Childhood Maltreatment: An Integrative Review

Abstract

Disordered eating behaviors and symptoms reflect an increased risk for eating disorders and are common, especially in emerging adult women aged 18-29. Studies have suggested an association between disordered eating symptoms and experiences of maltreatment in childhood. This integrative review summarized literature on the associations between disordered eating symptoms or behaviors, and the role of childhood maltreatment among emerging adult women. A search was conducted using PubMed, PsycINFO, and Scopus. Publications were included if they contained measures of disordered eating symptoms and experiences of childhood maltreatment in women ages 18-29. From the search of 399 articles, 13 articles met inclusion criteria. Twelve studies demonstrated an association between childhood maltreatment and disordered eating behaviors and symptoms among young adult women aged 18-29. Studies examined total maltreatment, childhood physical, sexual, emotional/verbal abuse and found associations with total Eating Disorder Examination Questionnaire scores, binge eating frequency, compensatory behaviors, shape and weight concerns, and disordered eating attitudes. Four studies examined mechanisms to explain the relationship between disordered eating behaviors and experiences of childhood maltreatment. This suggests that individuals who have experienced child maltreatment may need additional screening for disordered eating. Further exploration of the mechanisms by which disordered eating symptoms can occur in relation to experiences of childhood maltreatment is warranted.

Keywords: childhood maltreatment, disordered eating, review, women

Introduction

Eating disorders have the highest mortality rates (1.7-5.1 deaths per 1,000 person-years) of all mental health-related conditions (Arcelus et al., 2011; James et al., 2010) and are typically diagnosed between 18 and 21 years in the United States (Berg et al., 2009; Hudson et al., 2007). Prior to reaching thresholds for a formal diagnosis of an eating disorder, disordered eating behaviors and symptoms often occur at subclinical levels (Combs et al., 2013; Gauvin et al., 2009) and are more prevalent than clinical eating disorders, especially in women (Berg et al., 2009; Mintz & Betz, 1988). Disordered eating behaviors and symptoms are also predictive of clinical-level eating disorders, including a nine-fold increase in bulimia nervosa diagnosis and a 20-fold increase in anorexia nervosa diagnosis (Killen et al., 1994; Kotler et al., 2001). Because disordered eating behaviors can impact well-being and are a prevalent modifiable health risk for eating disorders (Nelson et al., 2008; Neumark-Sztainer et al., 2006), an understanding of how these behaviors develop is critical for the improvement of interventions to target reduction of these risky behaviors.

Disordered eating symptoms include a spectrum of eating behaviors and attitudes such as preoccupation with shape or weight, extreme caloric restriction, bingeing, or self-induced vomiting (American Psychiatric Association, 2013). Epidemiological studies find that 56% of 9th-grade women compared to 28% male peers and 57% of 12-grade girls compared to 31% male peers report disordered eating behavior (Croll et al., 2002) and that these behaviors can persist and increase during the emerging adult period (characterized as 18-29) (Arnett, Žukauskienė, et al., 2014; Goldschmidt et al., 2016). Studies of undergraduate women found 49% to 61% report disordered eating behavior

compared to 16% to 22% in men (Berg et al., 2009; Chatterton & Petrie, 2013; Mintz & Betz, 1988; Petrie et al., 2008). In addition to increased risk for clinical-level eating disorders (Hoerr et al., 2002; Shriver et al., 2016), disordered eating behaviors are also associated with weight gain (Delinsky & Wilson, 2008; Nagata et al., 2018), poor dietary intake (Larson et al., 2009), and depression and anxiety (Braun et al., 1994; Jacobi et al., 2004; Puccio et al., 2017).

In addition to being identified as a critical period for the development of disordered eating behavior and weight difficulties (Berg et al., 2009; Goldschmidt et al., 2016; Hunt & Eisenberg, 2010), emerging adulthood is characterized by frequent changes and transitions (e.g. housing), growing independence, and identity exploration (Arnett et al., 2014). It is also known a period of rapid brain development in areas associated with executive function, cognitive evaluation, and inhibition of impulsive, risky behaviors (Casey et al., 2008; Steinberg, 2008). These developmental process involves progressive lessening of impulsive behaviors counterbalanced by enhancements in goal-oriented behavior and with greater recognition of the negative consequences of behavior (Arnett, Taber-Thomas, & Pérez-Edgar, 2014). As a result of growing independence and autonomy, as well as continued brain development, the emerging adult period is an opportune time for identification of disordered eating and for intervention to prevent serious health complications.

Early childhood experiences can impact long-term health and behavior (Shonkoff et al., 2012). Compared to the general population, women with eating disorders are more likely report a history of childhood maltreatment (Smolak & Murnen, 2002), which includes experiences of harsh physical punishment, physical abuse, sexual abuse,

emotional abuse, neglect, and exposure to intimate partner violence (IPV) (Leeb, 2008). Furthermore, childhood maltreatment is associated with more than three times the odds of developing any kind of eating disorder (Afifi et al., 2017) and obesity (Danese & Tan, 2014). However, the relationships between experiences of childhood maltreatment and disordered eating behaviors is less clear and this body of literature has not yet been synthesized. The purpose of this integrative review is to synthesize the literature related to the association between disordered eating symptoms or behaviors among emerging adult women with experiences of maltreatment in childhood. An exploratory aim examined mechanisms that explain the association between disordered eating behaviors and symptoms in emerging adult women with experiences of childhood maltreatment.

Methods

Study Selection

This integrative review used the strategy outlined by Whittemore & Knafl (2005). Literature searches were conducted with the assistance of a university research librarian using National Library of Medicine Medical Subject Heading (MESH) terms and keywords related to childhood maltreatment and eating. The first author (JE) searched for literature published in English between January 2008 and January 2018 within the following databases: PubMed, PsycINFO, and Scopus. The terms child abuse, child maltreatment, disordered eating, disordered eating behavior, eating behavior were used for the search (**Table 2.1**). Studies were considered eligible for this review if they included empirical findings examining the association between childhood maltreatment (childhood physical, sexual, emotional abuse, or neglect) and disordered eating behavior in emerging adults aged 18-29. Among eligible studies, those included for analysis were articles with search terms found in the title or as keywords, subjects/participants with a

history of childhood maltreatment, and a measure of disordered eating behavior (**Figure 2.1**). Excluded from the review were articles not written in English, outcome measures that did not include a measure of disordered eating behavior and/or symptoms, and non-empirical papers including reviews, editorials, case reports, etc. and, studies involving animal research. Reference lists of all relevant publications were also reviewed for additional articles (Conn et al., 2003). As a framework for this integrative review, the search strategy utilized the guidelines provided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2009 Checklist.

Data Extraction and Assessment of Quality

With the search strategy described above, articles were extracted from academic databases into RefWorks for organization and categorization. The search strategy yielded 399 articles total. Following removal of duplicates ($n = 160$), and screening of title and abstract for measures of disordered eating and history of childhood maltreatment, 74 articles remained. Upon further review of the full article, articles were removed because they were outside of the average age (18-29 years), did not measure disordered eating behaviors, did not include a measure of childhood maltreatment, included samples $< 50\%$ women or samples of men, included people in treatment for eating disorders and/or with a diagnosis of an eating disorder, or only assessed weight status/body mass index. Of the 74 articles, 13 met the inclusion criteria and were evaluated for scientific rigor of study design, methods and analysis (**Figure 2.1**).

Study quality was assessed using the National Heart, Lung, and Blood Institute (NHLBI) Quality Assessment Tool for Observational Cohort and Cross-sectional Studies (NHLBI, 2014). This tool includes questions to assess bias, confounding, power, and strength of association between exposure and outcome of interest. The quality of the

study is determined after a careful evaluation of the methodology, validity of evidence, and potential bias presented in the results. Overall quality for each study is rated as “good” (low risk of bias, valid results), “fair” (some risk of bias, does not invalidate results), or “poor” (significant risk for bias, may invalidate results, significant methodological weaknesses).

Results

Study Design Characteristics

Of the 13 articles that were included in a synthesis of study results, 12 studies were cross-sectional and one study was prospective (Smyth et al., 2008). All studies utilized survey methodology. All study populations were mostly female (> 50%) and included participants recruited from university settings (n= 11) except for 2 studies that utilized national samples from the United States (Fuemmeler et al., 2009; Mason et al., 2015). The mean age of the study samples ranged from 18 to 26, however, one study described their sample as undergraduates without providing an average age (Dworkin et al., 2014). Of the studies that identified the racial/ethnic makeup of the samples (n= 12), 10 studies included predominantly participants who identified as White. The other two studies included 30% (Salwen et al., 2014) and 31% (Hymowitz et al., 2017) participants identifying as Asian. Study characteristics are summarized in **Table 2.2** and each individual study is summarized in the Table of Evidence (**Table 2.3**).

Summary of Results

Of the 13 studies reviewed, three studies looked at trauma as a composite measure and found that greater trauma was associated with greater Eating Disorder Examination Questionnaire (EDE-Q) scores (Smyth et al., 2008), increased number of eating problems (Stoltenberg et al., 2012), and greater number of disordered eating symptoms (Hasselle et

al., 2017). Only one study found that severe physical, sexual, and emotional abuse were each independently associated with increased risk for binge eating (physical risk ratio 1.44, sexual risk ratio 1.31, emotional risk ratio 1.52) and coping-motivated eating (physical abuse risk ratio 1.43, sexual abuse risk ratio 1.51, emotional abuse risk ratio 1.47) (Mason et al., 2015).

Two studies found childhood emotional abuse was associated with higher EDE-Q scores after controlling for sexual and physical abuse (Burns et al., 2012; Fischer et al., 2010). Childhood emotional abuse was also associated with binge eating, night eating syndrome, and unhealthy weight loss practices (Hymowitz et al., 2017; Salwen et al., 2015). Another study found that women with a history of physical abuse reported more problematic eating and skipping meals (Fuemmeler et al., 2009).

Only one study found no association between any type of childhood maltreatment and disordered eating behaviors (binging, purging, restraint, laxative misuse) when controlling for depression, anxiety, age, BMI, and socioeconomic status (Villarroel et al., 2012). This study also looked at the attitudes features of disordered eating and found that childhood physical abuse was associated with lower scores on the Weight Concern Subscale of the EDE-Q and the total EDE-Q score, but childhood sexual abuse was associated with higher score on the Weight Concern subscale. Another study found that childhood sexual abuse was associated with binge eating and compensatory behaviors (Dworkin et al., 2014) and a higher prevalence of eating problems (loss of control eating, being told they have an eating disorder) (Fuemmeler et al., 2009). One study did not find an association between childhood sexual abuse and EDE-Q score (Moulton et al., 2015).

Of the studies that identified specific disordered eating symptoms and behaviors, bingeing behavior and/or restrictive eating was the most often mentioned (n=5) (Burns et al., 2012; Dworkin et al., 2014; Hymowitz et al., 2017; Salwen et al., 2015; Smyth et al., 2008). Three studies also demonstrated a positive dose-response relationship such that the number of eating problems reported was related to the frequency/severity of the childhood maltreatment experiences (Fuemmeler et al., 2009; Smyth et al., 2008; Stoltenberg et al., 2012).

Mediators of Disordered Eating Behavior and Childhood Maltreatment

To explain the relationship between disordered eating symptoms and experiences of maltreatment in childhood, different types of mediators were explored in the literature – these included cognitive, psychological and genetic factors. Maladaptive emotions (Gilbert & Irons, 2009) like negative self-perception (Hymowitz et al., 2017) were found to mediate the relationship between disordered eating symptoms and experiences of childhood maltreatment. Similarly, impulsivity and compulsivity were found to be mediators, but compulsive tendencies more strongly explained this relationship between disordered eating and experiences of maltreatment in comparison to impulsive tendencies (Dworkin et al., 2014). Childhood emotional abuse, specifically, and disordered eating symptoms and binge eating was mediated by emotional dysregulation (Burns et al., 2012).

One study examined the influence of genetics, specifically, the serotonin transporter gene, a known regulator of eating behaviors (Ioakimidis et al., 2011). Women with lower expression alleles of the serotonin transporter gene that were exposed to greater levels of childhood trauma were more likely to have a greater number of eating problems and were more likely to endorse bingeing and purging behaviors compared to women with normal

and low expression of the serotonin transporter that experienced low levels of childhood trauma (Stoltenberg et al., 2012).

Measures of Disordered Eating Behavior

There were numerous methods utilized to measure disordered eating behaviors and included validated tools like the Eating Disorders Examination Questionnaire (n=6), Minnesota Eating Behavior Survey (n=1), Questionnaire of Eating and Weight Related Patterns (n=1), SCOFF Questionnaire on Eating Habits (n=1), Eating Disorder Diagnostic Scale (n=1), Eat Attitudes Test 26 (n=1), Night Eating Questionnaire (n=2), and unvalidated questions about problematic or disordered eating behavior (n=3) (Fuemmeler et al., 2009; Mason et al., 2015; Salwen et al., 2014) (**Table 2.2**).

Measures of Childhood Maltreatment

The Childhood Trauma Questionnaire (CTQ) was most often used to assess experiences of childhood maltreatment (n=3) (Burns et al., 2012; Fischer et al., 2010; Moulton et al., 2015). The Emotional Abuse subscale from the CTQ was used in one study (Hymowitz et al., 2017). Nine studies utilized other childhood maltreatment questionnaires or tools like Juvenile Victimization Questionnaire Screener (Hasselle et al., 2017), Early Memories of Warmth and Safeness Scale (Gois et al., 2018), Trauma Antecedent Questionnaire (Stoltenberg et al., 2012), Risky Families Questionnaire (Vartanian et al., 2014), Weight Related Abuse Questionnaire (Salwen et al., 2015), a combination of questionnaires (Fuemmeler et al., 2009; Mason et al., 2015), an unspecified trauma questionnaire (Smyth et al., 2008), and a measure adapted from a scale to assess childhood sexual abuse only (Dworkin et al., 2014).

Quality Assessment of Results

Few studies (n=4) were rated “good” quality using the NHLBI Quality Assessment Tool (**Table 2.2**). The remaining studies (n=9) were rated “fair” quality, and no studies were rated “poor” quality. Overall, the studies lacked adequate description of the recruitment and samples, relied on convenience samples, and did not provide estimated effect sizes and/or power analysis. There was also a lack of confounders accounted for in the analysis.

Discussion

Overview of Findings

Almost all studies in this literature review demonstrated that in emerging adult women, there is an association between disordered eating symptoms and experiences of maltreatment in childhood, however, the strength of that relationship is still unclear as many of the studies lacked effect sizes and results were often based on convenience samples. All studies were cross-sectional except for one study that found a relationship between the number of reported eating problems and severity of traumatic experiences in childhood, and increased disordered eating over the first semester of college (Smyth et al., 2008). This is an important finding that should be further characterized as it may be an opportunity for intervention.

Mental health issues like depression and post-traumatic stress disorder are often comorbid with disordered eating symptoms and experiences of maltreatment. Only one study accounted for depression, anxiety, age, BMI, and socioeconomic status, and found that there was no association between disordered eating symptoms and experiences of the sexual abuse form of childhood maltreatment (Villarroel et al., 2012). This type of maltreatment is less common compared to other forms (Merrick et al., 2018), so the study may not be powered to detect a difference. Importantly, while some studies assessed

depression and/or anxiety as potential covariates or mediators, no other studies accounted for socioeconomic status. Given that socioeconomic status may influence food insecurity, which has been shown to influence eating behavior and is associated with increased odds of binge eating behavior (Bruening et al., 2017) and higher body mass index (Oberle et al., 2019), it is important to consider socioeconomic status or food insecurity in future studies. Further, food insecurity can also be found in the university setting (Bruening et al., 2016), so this should be considered even in studies only examining students recruited from the university setting.

Mechanisms of Disordered Eating Behavior and Childhood Maltreatment

The literature exploring the mechanism by which experiences of childhood maltreatment is associated with disordered eating symptoms and behaviors is an important, but understudied area. It is likely that there is a psychological/mental health component to this relationship, as diagnoses of anxiety, depression, and post-traumatic stress disorder are often comorbid with clinical eating disorders (Hudson et al., 2007), and also associated with disordered eating symptoms (Lavender et al., 2015; Leehr et al., 2015), and childhood maltreatment (Briere & Jordan, 2009), independently. However, not all studies included within this review accounted for these mental health conditions, which may have influenced results. Notably, one study found female victims of childhood maltreatment were more likely to demonstrate disordered eating even when depression and PTSD were accounted for (Hasselle et al., 2017). This study did not apply a mediation analysis but included depression and PTSD as control variables. Future studies should collect data related depression, anxiety, and/or PTSD to determine how presence of these may influence the relationship between disordered eating and childhood maltreatment.

There is evidence to suggest that genetics account for about 50% of variance in disordered eating symptoms and eating disorders (Klump et al., 2001), while other studies suggest a combination of genetic and environmental influences (Mitchell et al., 2010). Despite this evidence, only one study examined any kind of genetic impact on disordered eating behavior and experiences of childhood maltreatment (Stoltenberg et al., 2012). Although the sample size was very ethnically homogenous to increase precision, the findings highlight a unique approach to determining the mechanism linking the association of disordered eating behaviors and experiences of maltreatment. In addition to genetic analysis of ethnically diverse people, future research should evaluate parental eating behaviors as this has been shown to influence child eating behaviors.

Other mediators examined in the literature range from cognitive dysregulation related to impulsivity/compulsivity (Dworkin et al., 2014). Previous studies hypothesized that bingeing and purging behavior in people with clinical eating disorders is the result of maladaptive affect regulation in response to trauma (Brewerton, 2007; Grilo & Masheb, 2001), however, maladaptive affect regulation has not been explored in disordered eating symptoms and childhood maltreatment is minimal as it is essential information for the development of intervention and screening tools. With the knowledge that a person has experienced childhood maltreatment, understanding the potential mechanism by which disordered eating develops can help better identify people at risk and inform intervention strategies.

Study Characteristics

The study populations were predominantly white/Caucasian and were recruited from universities/colleges (n= 13). These results demonstrate that there is a consistent relationship between disordered eating behaviors and experiences of childhood

maltreatment, but few studies (n=2) included participants outside the university settings (Burns et al., 2012; Stoltenberg et al., 2012). According to analysis from the Population Reference Bureau in 2017, approximately 50 percent of people aged 18-24 in the U.S. attend higher education or have an undergraduate degree (The Annie E. Casey Foundation, 2017), but that still leaves 50 percent of the population that is unaccounted for in studies that recruit only from universities.

The studies in this review were majority White participants, so the results cannot be generalized to other races and ethnicities. However, there is evidence that disordered eating behaviors are not unique to White women as studies show ethnic minority adolescents are at heightened risk for disordered eating compared to white adolescents (Austin et al., 2011; Croll et al., 2002) and Black women with experiences of childhood maltreatment report increased emotional eating or eating absence of physiological hunger compared to those without experiences of childhood maltreatment (Michopoulos et al., 2015). In female children, eating in the absence of hunger predicts future binge eating (Balantekin et al., 2017). Future studies should include more diverse samples.

Few studies included information related to socioeconomic status. One study found that when socioeconomic status was accounted for, there was no relationship between any type of childhood maltreatment and disordered eating behaviors. Food insecurity is a proxy for socioeconomic status is prevalent in university students (Bruening et al., 2016) and is linked to heightened exposure to violence (Chilton et al., 2014). Studies show food insecurity may influence eating behavior and is associated with increased odds of binge eating behavior (Bruening et al., 2017) and higher body mass index (Oberle et al., 2019). Previous studies show that childhood maltreatment is strongly associated with poverty

and low-academic achievement (Gilbert & Irons, 2009) and Black and Latino populations are disproportionately exposed to trauma compared to White people (Smyth et al., 2008). Therefore, by limiting study populations to white undergraduate students, the strength of the association between experiences of childhood maltreatment and disordered eating is potentially underestimated. This highlights a serious gap in the literature that warrants further study.

A methodological consideration not found in this literature search, but found in the eating disorder literature, is the utilization of brain imaging techniques to examine individual differences in the brain of people that report disordered eating behavior compared to those that do not report disordered eating behavior. Evidence suggests that people with experiences of maltreatment in childhood demonstrate changes in brain function and connectivity (Teicher et al., 2016); this may contribute to increased risk for disordered eating symptoms possibly as a result of altered regulatory and reward systems in the brain.

Measures of Eating Behavior

The measurement tools utilized to study disordered eating symptoms and behaviors associated with childhood maltreatment ranged from validated tools to screening tools like the Eating Disorder Examination Questionnaire (Fairburn & Beglin, 1994), to unvalidated questions, thus adding a challenge to the generalization of the findings across studies. Although the tools utilized often measured similar behaviors like binge eating behavior, emotional eating behaviors, and behaviors related to unhealthy weight loss practices, for example, the way these questions were employed varied. It is difficult to generalize a specific behavior without a comprehensive tool or standardized measure that examines all disordered eating and the associated outcomes. Nevertheless, this review

does highlight some patterns in the relationship between childhood maltreatment and disordered eating behaviors.

Limitations

This review is not without limitations. First, articles were collected from the previous 10 years and limited to those available in full-text format. One reviewer was involved in the literature search, organization, and screening from a selected number of databases. To increase rigor of this review, the examination chair was consulted on several occasions for confirmation of inclusion and exclusion of articles. All studies were cross sectional, except one prospective study that included assessment at 2 time periods, so the extent that disordered eating behaviors persist throughout early adulthood is unclear.

Future Directions

Despite the consistent evidence favoring a relationship between disordered eating behavior in emerging adult women and experiences of maltreatment in childhood, there remain many gaps in the literature. Future studies should collect data related to environmental factors (socioeconomic status, parental eating patterns, family medical history). Recruitment should target participants identifying as non-white/Caucasian and from populations outside of university settings. Measures of eating behavior should be standardized to improve characterization and should be included in longitudinal studies and/or at primary care health assessments in order to gain a better understanding of how eating behavior develops over time. Finally, examining how the developing brain is implicated in disordered eating behavior will aid in identification and may provide guidance for targeted interventions.

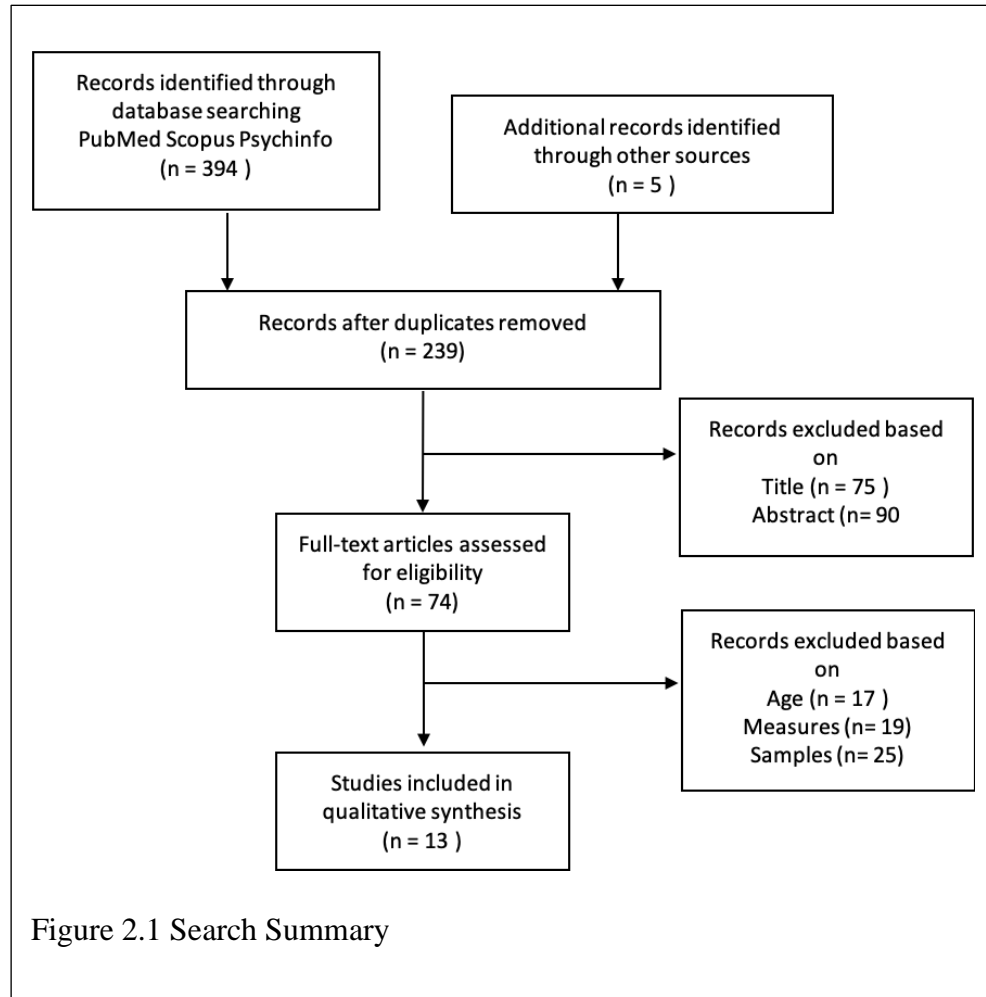


Figure 2.1 Search Summary

Table 2.1 Search Strategy and Terms Used			
Database	PubMed	PsychINFO	Scopus
Search terms used	Child Abuse Domestic Violence Family Violence Eating Food	Child Abuse Child Maltreatment Eating Behavior Disordered Eating	Child abuse Child maltreatment Disordered eating
Search Code	((("Adult Survivors of Child Abuse"[Mesh] OR "Child Abuse"[Mesh]) OR "Domestic Violence"[Mesh] OR "family violence" OR "domestic violence" OR "child abuse")) AND (("Eating"[Mesh]) OR "Food"[Mesh] OR eating[title] OR food[title])	mainsubject(child abuse) OR mainsubject(child* maltreatment) AND mainsubject(eating behavior) OR mainsubject(disordered eating) NOT mainsubject(eating disorder)	(TITLE-ABS-KEY (child AND maltreatment) AND TITLE-ABS-KEY (disordered AND eating))
Filters applied (if applicable)	Published in the last 10 years; English	2008-2018 Peer-reviewed Female English language Young Adulthood (18-29 Yrs)	None applied

Table 2.2 Study Characteristics		
	Number of Studies (n=)	% of Studies
<i>>50% identified as Caucasian/white</i>	11	84.6
<i>College Students</i>	10	66.7
<i>Measures of eating behavior</i>		
Eating Attitudes Test 26	1	6.7
Eating Disorder Examination-Questionnaire	5	40
Night Eating Questionnaire	2	13.3
Questionnaire on Eating and Weight Related Patterns	2	13.3
SCOFF Questionnaire on Eating Patterns	1	6.7
<i>Mediators</i>		
Anxiety, depression, PTSD	4	20
Impulsivity/Compulsivity	1	5
Negative self-perception/self-criticism	2	10
<i>Quality Type</i>		
Good	4	31
Fair	9	69
Poor	0	0

Table 2.3 Table of Evidence					
Author & Publication Year	Purpose/Objective	Study Design/Methods & Measures	Sample Size & Population Characteristics	Relevant Findings	Study Quality
Burns et al. 2012	Examine the relationship of child maltreatment to emotion dysregulation and subsequent eating pathology.	Cross-sectional Survey data, Structural equation modeling Difficulties in Emotion Regulation Scale EDE-Q ₂	1,301 women Age 18- 22 White 77.7% Women 100%	Childhood physical and emotional abuse were significantly associated with bingeing behavior. Emotional abuse also correlated with overall EDE-Q. Emotion dysregulation was correlated with eating disorder symptoms and partially mediated the association between emotional abuse and binge eating and EDE-Q score.	Fair
Dworkin et al. 2014	Examine impulsivity and compulsivity as mediators of the relationship between childhood sexual abuse victimization and distinct disordered eating behaviors.	Cross-sectional Survey Structural equation modeling Minnesota Eating Behavior Survey (MEBS)	649 undergraduates Mean age: Not reported Women 66.6% White 84.3%	17.87% of the women reported sexual abuse, women had statistically significant MEBS scores, bingeing, and compensatory behaviors compared to males. Binge eating and compensatory behaviors were mediated by impulsivity and compulsivity in people with experiences of sexual abuse. Authors suggest compulsive tendencies play a greater role in disordered eating behaviors.	Fair
Fischer et al. 2010	Examine the effect of recent adult sexual assault on current eating disorder symptoms when controlling for the	Cross sectional Survey EDE-Q ₂	489 undergraduate women Modal age 18 Women 100%	Linear regression of scores on the Global Scale of the EDE-Q were regressed onto centered scores on the sexual, physical, and emotional abuse scales of the Childhood Trauma Questionnaire.	Fair

	effects of multiple forms of childhood abuse.		White 75.6%	Emotional abuse was the only significant predictor of current disordered eating symptoms.	
Fuemmeler et al. 2009	Examine the relationship between childhood abuse and obesity in young adulthood in a large representative U.S. sample.	Cross sectional Secondary analysis of the National Longitudinal Study of Adolescent Health (Add Health) Height/weight measured Disordered eating Problematic eating behaviors (feeling afraid to eat, and physician indicated eating disorder)	4099 women Mean age (SD) 22 (1.8) Women 100% White 74.6%	Women that reported sexual abuse reported greater prevalence of problematic eating behaviors. Childhood physical abuse was associated with skipping meals. Childhood sexual abuse and childhood physical abuse was associated with being told they have an eating disorder or being afraid to eat because they may lose control. No association between childhood abuse and obesity or overweight was observed in sample.	Fair
Gois et al., 2018	Examine the mediator roles of self-criticism and shame in the link between early emotional experiences and the engagement in disordered eating.	Cross sectional Secondary data, path analysis EDE-Q ₂	552 participants from general population Mean age (SD) 25.47 (5.86) Women 100% White 100%	The absence of early positive emotional experiences was associated with disordered eating behaviors, through an increased perception of being negatively perceived as inferior or unattractive by others, self-critical attitudes, and body image-focused shame. Shame and self-criticism are defensive mechanisms associated with early threatening emotional experiences, which may trigger disordered eating behaviours.	Fair

Hasselle et al. 2017	Examine the relationship between childhood polyvictimization and disordered eating symptoms (DES) in emerging adults.	Cross sectional Hierarchical multiple regression SCOFF	288 college students Women 71.8% Mean age (SD) 19.19(1.40) White 66.3% Black/African American 17.4%	Women participants that experienced childhood victimization were more likely to demonstrate DES. Women participants were also more likely to demonstrate DES even when depression and PTSD were accounted for.	Fair
Hymowitz et al. 2017	Evaluate a model of disordered eating and weight difficulties in which negative self-perception mediate the relationship between emotional abuse and disordered eating and disordered eating predicts body mass index in a population of emerging adults.	Cross sectional Online and in person questionnaire Questionnaire of Eating and Weight Patterns-Revised Night Eating Questionnaire Weight and Lifestyle Inventory	598 undergraduates Women 60.4% Age mean (SD) 19.51 (1.54) Caucasian 45.7% Asian 31.1% Hispanic 8.4% African-American 6.5% bi-racial or other 8.4%	Negative self-perception mediates the association between emotional abuse and disordered eating (binge eating, night-eating syndrome, and unhealthy weight control). In turn, disordered eating predicts higher BMI. High levels of emotional abuse, are more likely to meet criteria for night-eating syndrome or binge eating, and emotional abuse has a moderate to high level of specificity as a predictor of binge eating and night eating syndrome.	Fair
Mason et al., 2015	Investigate whether two stress-related overweight behaviors—binge eating and coping motivated eating—explain childhood abuse associations with weight status in young women.	Cross sectional Growing Up Today Study Marginal structural models	4377 women “Majority white” 22 to 29 years	Women with severe physical, sexual, and emotional abuse had early adult body mass indexes (BMIs) that were 0.74 kg/m ² (95% confidence interval [CI]: 0.15–1.33), 0.69 (95% CI: –0.46 to 1.83), and 0.85 (95% CI: 0.24–1.45) kg/m ² higher, respectively, than those without abuse. Adjustment for coping-motivated eating attenuated the excess BMI associated with severe physical abuse.	Good

Moulton et al., 2014	Investigate whether childhood trauma is indirectly associated with eating psychopathology through mediation by dissociation and/or emotion dysregulation.	Cross sectional survey Multiple mediation analysis EDE-Q ₂	142 British undergraduates Age mean (SD) 21.06 (4.84) Did not report race/ethnicity of sample	Emotion regulation and dissociation significantly mediate the relationship between childhood trauma and eating psychopathology.	Fair
Salwen et al., 2015	Evaluate the relationship between weight-related abuse (WRA) and the development of disordered eating. The perceived emotional impact of WRA mediates the relationship between WRA and current disordered eating.	Cross-sectional survey Night Eating Questionnaire POTS Questionnaire of Eating and Weight Problems-Revised Unhealthy weight control Three Factor Eating Questionnaire-18R Weight Related Abuse Questionnaire	383 undergraduates Mean age (SD) 19.36 (1.59) range 18-25 Women 56.4% White 48.7% Asian 30.3%	Women reported significantly more emotional eating than men. All other differences were not significant. Verbal weight related abuse was significantly associated with disordered eating after controlling for childhood verbal abuse and weight related teasing. The perceived emotional impact of verbal weight related abuse mediates the relationship between verbal weight related abuse and disordered eating.	Fair
Smyth et al., 2008	Examine whether reported history of adverse life events predicted eating disturbance upon college entry and prospective changes over the first semester of college.	Observational cohort EDE-Q ₂	249 First year college students, 219 at follow up Age 18-19 Women 55% White 96.3%	Restrictive eating was significantly more common when a greater number of traumas were reported, total trauma severity was rated higher, and more severe violent trauma was reported. Binging/purging behaviors were reported more often for individuals who indicated a greater number of traumatic experiences, greater total trauma severity, and a higher severity rating violent trauma.	Good

Stoltenberg et al., 2012	Investigate a potential interaction between the triallelic polymorphism of the serotonin transporter gene (SLC6A4) promoter and the experience of childhood trauma on the number of problem eating behaviors.	Cross sectional survey Linear modeling EAT-26 ¹	439 women, university students Mean age 22.49 (SD 6.12) 85% aged 18-25 Female 100% White 100%	Women carrying lower expression alleles who reported being exposed to higher levels of childhood trauma reported a significantly higher mean number of eating problems. Women in the high risk group were also more likely to report having been treated for an eating disorder and endorse bulimic specific symptoms. Low expressions of the 5-HTTLPR triallelic genotype was also associated with greater number of eating problems. Low levels of gene expression with high levels of childhood trauma was not associated with elevated scores on BIS-11.	Fair
Vartanian et al., 2014	Examine the mechanisms underlying the association between early adverse experiences and disordered eating.	Structural equation modeling Body shape Questionnaire EDE-Q ₂	748 undergraduates private university, Northeastern U.S. Mean age (SD) 19.75 (1.27) Female 100% White 70.3%	Greater body dissatisfaction predicted more disordered eating (binging, purging, restricting behaviors). Early adversity predicted decreased intrapersonal resources (self-esteem, personal growth), which was associated increased body dissatisfaction.	Good
Villaroel et al., 2011	Assess the relationship between childhood sexual and physical abuse and key attitudinal and behavioral aspects of eating disorders.	Cross sectional, survey EDE-Q ₂	708 undergraduates, Spanish public university Mean Age (SD) 22.0 (2.7) Caucasian 94.3%	After adjusting for depressive and anxiety symptoms, age, BMI and socioeconomic status, they found no significant association between childhood sexual abuse and the behavioral features of eating disorders.	Good

¹Eating Attitudes Test- 26; ²Eating Disorder Examination- Questionnaire

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Chapter 3

An fMRI Study of the Neural Substrates of Hedonic Hunger in Emerging Adult Women and the Moderating Role of Childhood Maltreatment

Abstract

Obesity is a prevalent public health issue and current research seeks to identify modifiable risk factors to enhance treatment and prevention. Studies suggest people with neural vulnerabilities to rewarding stimuli like food, may be more likely to eat in the absence of hunger. Further, experiences of childhood maltreatment may contribute to increased vulnerability due to the impact of toxic stress on the brain. The purpose of this study was to examine the neural correlates of hedonic hunger as measured by the Power of Food Scale score. An exploratory aim examined the moderating effects of experiences of childhood maltreatment. A secondary analysis of the BUFR2 Study was conducted. The BUFR2 study explores the relationship between sexual risk behaviors in emerging adult women and response to appetitive cues (food, sexual, aversive). Analysis of the data revealed people with greater hedonic hunger had decreased neural activation to novel food cues in areas associated with interoceptive awareness and executive function. We also found that greater hedonic hunger was associated with increased activation in neural areas associated with interoceptive awareness. Together these results suggest that people with higher levels of hedonic hunger do not have the expected reduction in the brain's response to repeated stimuli and may contribute to behaviors associated with obesity. Further exploration of the temporal dynamics of neural response to food cues and hedonic hunger is warranted.

Introduction

Obesity, defined as a body mass index (BMI) greater than 30 kg/m² (World Health Organization, 2018), is a chronic relapsing disease that results in significant morbidity and mortality (Bray et al., 2017). Of significant concern is the growing number of emerging adults (18-29 years) with obesity, as this group has greater increases in obesity than any other age groups in the United States (Ogden et al., 2017) and is a period known for significant weight gain (Zheng et al., 2017). There are also notable differences based on gender and socioeconomic status. The prevalence of obesity in women decreases as income increases (45.2% to 29.7%), while there are no differences in men (Ogden et al., 2017). This is of particular concern in urban environments where cheap, highly processed and nutrient poor foods are often easily accessible and associated with excess energy intake, weight gain, and obesity (Blundell & Finlayson, 2004; Johnson & Wardle, 2014). However, there are individual differences in how people respond to these obesogenic environments. Therefore, understanding why some people are more sensitive to overeating in obesogenic environments requires further exploration. To address this research question it is necessary explore the neural underpinnings of eating and its contribution to the development and maintenance of obesity.

The two primary factors that regulate food intake are homeostatic, physiological hunger and pleasure-based, hedonic-driven hunger. Physiologic hunger occurs when the body is acutely deprived of sufficient calories needed to maintain homeostatic function. Hedonic hunger occurs in the absence of acute caloric needs and is associated with pleasure-based eating (Lowe & Butryn, 2007). Current neurobiological research suggests that palatable food activates the dopaminergic pathway to release dopamine, which

stimulates areas of the brain that signal reward (K. C. Berridge & Robinson, 1998; Schultz, 2006; Nora D Volkow et al., 2017). Importantly, cues (sights, sounds, smell, etc.) that signal the arrival of food can themselves acquire the ability to trigger dopamine release, acting as ‘incentive’ stimuli that trigger food desire, “craving” and hedonic hunger (Berridge & Robinson, 1998). People with increased sensitivity to environmental food cues may have greater motivation to seek out food and be more likely to overeat (Ferriday & Brunstrom, 2011; Wang et al., 2004), thus contributing to increased risk for overweight and obesity (Berridge, 2009; Polivy et al., 2008). However, the evidence to support this hypothesis is still limited, therefore, it is vital to further understand the neural mechanisms underlying behavioral responses to food cues that may contribute to hedonic hunger and overeating.

The Power of Food Scale (PFS) was developed to measure hedonic hunger or an individual’s motivation to consume food in an obesogenic environment (Lowe & Butryn, 2009; Lowe et al., 2009). Neuroimaging studies reveal that in response to food cues, higher PFS scores are associated with increased connectivity in the visual cortex (Bullins et al., 2013), increased connectivity in the cerebellum and surrounding areas associated with behavior (Rejeski et al., 2012), and increased activation in the insular cortex (Yoshikawa et al., 2013, 2014) and post central gyrus, which are associated with the somatosensory properties of food (Burger et al., 2016). Another study demonstrated that higher PFS scores were associated with decreased activation in inhibitory regions in response to high calorie foods compared to low calorie foods (Jensen et al., 2016). Only one of these studies looked at emerging adult women specifically, but these participants were actively participating in a weight gain prevention program (Burger et al., 2016).

Furthermore, because the obesogenic environment is filled with food-related conditioned stimuli, repeated presentation of cues may provide a sensitive – and more realistic – paradigm for understanding the motivational response to food cues and for revealing the neural correlates of hedonic hunger.

In economically disadvantaged urban environments, which tend to have a greater minority population, greater rates of obesity, and more food insecurity, early experiences of adversity, like childhood maltreatment (e.g., emotional, physical, and sexual abuse before age 18) are more common (Herrenkohl & Herrenkohl, 2007). These experiences are associated with depression (Briere & Jordan, 2009), can impact brain development in areas associated with reward (Novick et al., 2018), and may contribute to increased risk for obesity (Danese & Tan, 2014). One previous study examined the neural response to food cues in adolescents with low and high experiences of trauma and found there was no difference between the groups in response to food cues (Else et al., 2015). Although there is no difference between experiences of high and low trauma, it is possible that childhood maltreatment may influence the strength of the relationship between the neural response to food cues and the motivation to consume food.

The primary aim of this study was to examine the association of hedonic hunger, as measured by the Power of Food Scale, with the neural response to food cues measured by fMRI in emerging adult women. We hypothesized that increased neural response to food cues in areas associated with reward would be associated with higher Power of Food Scale scores. An additional exploratory aim of this study examines how experiences of childhood maltreatment may better explain the correlation between the neural response to food cues and the Power of Food Scale in reward-associated areas.

Methods

Study Design

A secondary data analysis of the Behavior in Urban Female Risk Study (BUFR 2) was performed. The parent study (BUFR 2) was designed to examine the neural response to neutral and evocative cues (food, sex, aversive) in emerging adult women (18-24) with varying degrees of risk for sexually transmitted infections (STIs)/HIV infection.

Participants (n= 60) were recruited from a federally supported Title X (serving low income individuals) family planning clinic and from a nearby university, both of which were located in a large urban area in the northeast United States. Subjects participated in two sessions, typically scheduled on consecutive days. In the first session, participants completed informed consent, surveys, and demographic health information via pen and paper. For sensitive topics like intimate partner violence and adverse childhood experiences, Audio Computer Assisted Self-Interviewing (ACASI) was utilized for data collection in order to increase the accuracy of self-report data (Morrison-Beedy et al., 2006). In the second session, a functional magnetic resonance imaging (fMRI) scan was completed while participants performed selected tasks on a computer. Due to the nature of fMRI, participants were excluded if they had a history of a seizure disorder, were greater than approximately 300 pounds, or were pregnant. Following completion of the study, participants were compensated 105 dollars. This study was approved by the institutional review board.

Sample and Setting

Study participants were a convenience sample selected from the BUFR 2 study. Inclusion criteria for this analysis were: age 18-24 years, female gender, a Power of Food Scale score and an fMRI scan that included the cue reactivity task. From the original sample of

60 subjects, 52 met inclusion criteria. Subjects (n= 8) were excluded due to incomplete fMRI data.

Measures

Hedonic Hunger. Hedonic hunger was assessed by the Power of Food Scale (Capelleri et al., 2009). This scale is a self-report measure of the motivational reward value for palatable foods and assesses the degree to which a person thinks about food in the absence of physiologic hunger. This scale contains 15 items with Likert-type response choices (1 = *do not agree at all*, 5 = *strongly agree*). Higher scores indicate increased motivational sensitivity to palatable foods. This measure has demonstrated high internal consistency ($\alpha = 0.80\text{--}0.91$) (Cappelleri et al., 2009) and good test–retest reliability (0.77) (Lowe et al., 2009). The alpha for the current sample was 0.93.

Child Maltreatment. The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1997) is a self-report measure that includes 28 items to rate the severity of emotional abuse and neglect, physical abuse and neglect and sexual abuse. Items are rated on a 5-point scale ranging from 1 (never true) to 5 (very often true). The sum of the five items for each subscale ranges from 5 to 25. Moderate to severe abuse is indicated by scores 13-15 for emotional abuse, 10-12 for physical abuse, 8-12 for emotional abuse, 15-17 for emotional neglect, and 10-12 for physical neglect. Cronbach's alpha for the factors ranged from 0.79 to 0.94, indicating high internal consistency. The CTQ also demonstrated good test-retest reliability over a 2- to 6-month interval (intraclass correlation = 0.88) (Bernstein et al., 1997). The Cronbach's alpha for this sample is 0.766.

Food Insecurity. Three questions assessed food insecurity: 1) Do you ever have to make choices between spending money on food or spending money on other needs? (i.e. medication, utilities, rent, etc.); 2) Do you ever worry whether your food will run out

before you have money to buy more?; 3) In the last three months, about how many times have you skipped a meal or cut the size of your meal because you do not have enough money for food? For question 1 and 2 the options are 1 (never), 2 (sometimes), 3 (often). For question 3, response options ranged 1 to 5 (1= 0 skipped meals, 2= 2-5, 3= 6-10, 4= 11-20, 5 >20). Question 1 and 2 have a 97% sensitivity for food insecurity indicated by scores of often or sometimes true compared to never true (Hager et al., 2010). The range of scores possible is 3 to 11. A total score >3 indicated food insecurity for this sample. The Cronbach's alpha for this sample was 0.779.

Depression. The Center for Epidemiological Studies-Depression (CES-D) (Radloff, 1977), is a 20-item measure that asks participants to rate how often over the past week if they experienced symptoms associated with depression, such as restless sleep, poor appetite, and feeling lonely. Response options range from 0 to 3 for each item (0 = Rarely or None of the Time, 1 = Some or Little of the Time, 2 = Moderately or Much of the time, 3 = Most or Almost All the Time). Scores range from 0 to 60, with high scores indicating greater depressive symptoms. It has a reliability of 0.85 across diverse populations (Lewinsohn, Seeley, Roberts, & Allen, 1997). The Cronbach's alpha for this sample was 0.84.

Demographic and Health Variables. Participants were assessed for age, current education status, race (Caucasian/White, African American/Black, Native American, Asian/Pacific Islander, Other), and ethnicity (Hispanic/Latino).

Imaging. Participants underwent a blood-oxygen-level dependent (BOLD) fMRI scan. The imaging center contains a Siemens 3 Tesla (Trio) research-dedicated magnet, an 8-channel head-coil, an LCD projector for stimulus presentation, air-conducting earphones,

and a fiber optic response pad. Mirrors, attached to the head coil, are adjusted so that participants can focus attention on stimuli and instructions projected on a computer screen. Prior to the functional scans, a 3-minute localizer scan and a T1-weighted high-resolution resting scan (5 minute) was acquired. For functional scans: T2*-weighted BOLD images were obtained with single shot gradient echo planar imaging sequence (field of view = 192 mm, matrix 64x64, TR=2 sec, TE=30ms, flip angle=80). In the scanner, participants were shown 500 millisecond evocative (food, sexual/romantic, and aversive) and neutral cues (**Figure 3.1**). Target stimuli were interspersed with gray screens with a single crosshair presented at a random duration between 1,000 millisecond and 2,000 millisecond, with an average of approximately 1,500 millisecond.

The task consisted of two rounds of cue presentation. Twenty-four novel cues from each category were presented in pseudorandom order, *followed by repeated presentation of those same cues*. The food cues (e.g., images of desserts, pasta, French fries, “highly palatable foods”), neutral cues (household or office objects; outdoor scenes) were from laboratory archives. The aversive cues and more than half of the sexual/romantic cues were selected from the top quartile (e.g., “most unpleasant” and “most pleasant”, respectively) of the International Affective Picture System (Lang et al., 1999). The remainder of the sexual/romantic cues were specifically generated to reflect greater racial diversity. Following observation of the food cues, participants completed a computerized task to rate the cues 0-9, 0 being the most unpleasant and 9 being the most pleasant.

Data Analysis

Descriptive statistics (mean, standard deviation, median, range, and percentiles) were produced to describe the sample characteristics. All data were closely examined for systemic patterns of missingness. Bivariate correlations were calculated between

variables of interest and potential confounding variables. P-values of 0.05 or less were considered to indicate statistical significance. All statistical analyses were performed using SPSS software version 25.0 (IBM Corp., NY).

fMRI data were preprocessed using a standard Statistical Parametric Mapping 12 (SPM 12; Wellcome Department of Cognitive Neurology, 2014) batch mode scripts modified from ASLtbx. Basic steps, including slice-timing correction, motion correction, temporal filtering, spatial smoothing, and registration to the Montreal Neurological Institute (MNI) standard brain, were processed using a standard SPM12 pipeline. The motion time courses were further removed from fMRI data using simple regression. Spatial smoothing was performed with an 8 mm³ full-width half-maximum Gaussian kernel.

Statistical analyses used a general linear model with a canonical hemodynamic response function with both the first (time) and the second (dispersion) derivatives using SPM 12 and MATLAB (Mathworks, 2019a). Three first-level contrasts [(Food – Neutral) in first and second halves of the task (*Food1 - Neutral1*, *Food2 – Neutral2*), food, second half minus food, first half (*Food2 – Food1*)] were defined to assess the effects of repeated cue presentation correlated with the Power of Food Scale total score, using a mask of the whole brain. Clusters were considered significant at $p < 0.01$, cluster-corrected with Analysis of Functional Neuroimages (AFNI version 20.0.19) program 3dClustSim and its autocorrelation feature (Statistical and Scientific Computing Core, 2019) to account for non-normal distribution of fMRI data (Cox et al., 2017). Parameter estimates were extracted from significant clusters within regions of interest to be used in the moderation analysis. To produce images, coordinates and t-values were extracted from SPM and

regions were identified using MNI (nearest grey matter) Atlas in Mango. The cue-rater was also correlated with the neural response to the food cues across the whole task.

To explore the impact of childhood maltreatment on the relationship between neural responses to food cues and hedonic hunger, the Baron and Kenny (1986) moderation analysis was applied. A multiple linear regression was conducted. The independent variables of the regression were neural response to food cues and childhood maltreatment and the interaction between neural response to food cues and childhood maltreatment. The interaction was created by multiplying the beta values from selected regions and childhood maltreatment together after both had been centered to have a mean of 0. The dependent variable of the regression was hedonic hunger as measured by the Power of Food Scale.

Results

Demographic characteristics of primary study variables are reported in **Table 3.1**. The sample consisted of 52 women with an average age of 20.9 (standard deviation: 2.5) years. Sixty percent (60%) of the sample self-identified as Black or African American. A majority of participants were not students (62% v. 38%). BMI data was collected for 47 participants and 60% of the sample had a normal BMI. The depression scores (CES-D) averaged 17 (s.d. 11) and the maltreatment scores (CTQ) averaged 39 (s.d. 16). The Power of Food Scale total score averaged 43(s.d. 15). The range of scores for food insecurity questions is 3-10 (average 4.4, s.d. 1.8).

The Power of Food Scale score correlated with food insecurity question 2 which asks, ‘Do you ever worry whether your food will run out before you have money to buy more?’ ($p < .05$). Maltreatment correlated with depression ($p < .01$) and food insecurity question

2 ($p < .01$). BMI did not correlate with any variables in this study. Correlations results are found in **Table 3.2**. There was no significant difference between Power of Food Scale scores and participants with and without Food Insecurity.

We sought to explore the relationship between the neural response to food cues and the Power of Food Scale. fMRI results showing changes in BOLD signal in response to food cues correlated with the Power of Food Scale total score in the 4 different analyses (whole time series, Food1-Neutral1, Food2- Neutral 2, Food2-Food1) are presented in **Figure 3.2**. The regions, coordinates, cluster size, and t-values are presented in **Table 3.3**. Across the whole time series, there were no significant clusters present. The neural response comparing Food1 – Neutral1 demonstrated decreased activation with respect to the Power of Food Scale scores in the cingulate gyrus, insula, and medial frontal gyrus in the negative direction, indicating an inverse relationship (**Figure 3.2a**). The neural response comparing Food2 – Neutral2 revealed increased activation in the post central gyrus (**Figure 3.2b**). Finally, the neural response comparing Food2 – Food1 revealed increased activation in the insula (**Figure 3.2c**).

To explore the moderating effect of experiences of childhood maltreatment, beta values were extracted from one significant cluster in each analysis, Food1-Neutral1, Food2- Neutral2, and Food2- Food1 for a total of 3 sets of beta values (**Figure 3.4**). Two regression models were created for each set of beta values (Food1 – Neutral1, Food2 – Neutral2, Food2 – Food1; Table 3.5) to test the effect of the moderating variable on the regression. Model 1 contained the two independent variables, beta value and CTQ score, and was significant in each of the 3 conditions (Table 3.5a $p < .001$; Table 3.5b $p < .003$; Table 3.5c $p < .003$). However, when adding the moderating variable (beta value*CTQ)

to each model, the interaction did not significantly account for the variance therefore, a moderation is not supported.

Discussion

In this study, we explored the relationship between the neural response to food cues and the Power of Food Scale score and found no significant correlation between the neural response to food cues and the Power of Food Scale score across the whole task. Our results may be different from previous findings of increased neural responding in somatosensory regions and decreased neural responding in the prefrontal cortex due to differing methods and study samples. Our study used palatable food images, while other studies used a food word (Bullins et al., 2013; Rejeski et al., 2012), and high and low calorie food cues (Jensen et al., 2016). The current study examined emerging adult women and utilized fMRI, while other studies looked at adolescents aged 14-20 (Jensen et al., 2016) or emerging adult men and utilized magnetoencephalography (Yoshikawa et al., 2013, 2014).

Although there was no relationship across the whole time series in both comparisons, we did find a significant relationship when examining the *temporal dynamics* of the neural response to food cues. Specifically, when observing food cues in the first half of the task, an inverse relationship between the neural response and the Power of Food Scale score. Higher Power of Food Scale scores were correlated with decreased neural response to food cues in the cingulate, insula, and the medial frontal gyrus and nodes in the dorsolateral prefrontal cortex. In the second half of the task, increased neural response in the post central gyrus was also associated with higher Power of Food Scale scores. Lastly, when comparing the neural response in first half to the second half, *increased*

activation in the insula and the anterior cingulate were also associated with higher Power of Food Scale scores. To our knowledge, no previous studies have looked at the temporal dynamics of neural response to food cues and the Power of Food Scale score.

Observation of a novel cue results in heightened neural response that decreases after repeated exposure to the same cue (Rankin et al., 2009). This response can be the result of habituation that is a reduction in responding (Epstein et al., 2009), or extinction that is a reduction in responding due to the omission of the primary reinforcer (e.g., food) (Bouton, 2011). The reduced response is the expected response, however, in certain psychiatric and neurological disorders, this reduced response does not occur and repeated cues elicit sustained excitation in the brain (Blackford et al., 2013; Dudas et al., 2017; Kleinhans et al., 2009). Our results suggest that people with greater Power of Food Scale scores may be less responsive to novel cues, but more responsive after repeated cues and this may contribute to greater motivation to consume food and increased risk for hedonic eating. Although there is an abundant of evidence supporting increased sensitivity to food cues in people with obesity compared to lean or normal weight individuals (Pursey et al., 2014), there is limited to no evidence on the neural response to *repeated* food cues. One study found people with obesity showed greater attentional bias to food cues (Castellanos et al., 2009) while another study found greater salivation in women with obesity compared to women without obesity (Epstein et al., 1996). Together these results suggest increased sensitivity to reward; however, it is unclear how these are related to neural response.

Our exploratory aim examining the moderating effect of maltreatment on the relationship between the neural response to food cues and the Power of Food Scale score

was not supported. This could be for several reasons. Previous studies show that there are differences in neural response based on high and low experiences of maltreatment (Elsey et al., 2015), however, our study was focused on the cumulative effects of maltreatment. Furthermore, there is some evidence that different types of maltreatment (e.g., physical, sexual, emotional abuse v. neglect) can differentially impact neural responding to cues (Puetz et al., 2019), however it is unclear how different types of maltreatment impact neural responding to food cues. This is a future avenue for research.

Finally, the overall average of food insecurity in our sample indicated that there was some degree of food insecurity within the sample (average score 4.7), with 28 participants endorsing that they sometimes must choose between food and other things or concern about running out of food (Hager et al., 2010), but it is unclear how this may have contributed to the results. There is no significant difference between the Power of Food Scale scores and participants with and without Food Insecurity, however, our sample size is not powered to draw any definitive conclusions. A recent study found that connectivity in the mesolimbic region was decreased in individuals that experienced food insecurity compared to those without (Ikuta & Holben, 2019). Another study found cognitive decline in older adults with food insecurity compared to older adults without (Frith & Loprinzi, 2018), although the specific areas of decline are unknown because fMRI was not utilized. Combined, these results suggest that food insecurity does impact cognitive function in areas associated with reward processing, but it is unclear if this was true for our group of women.

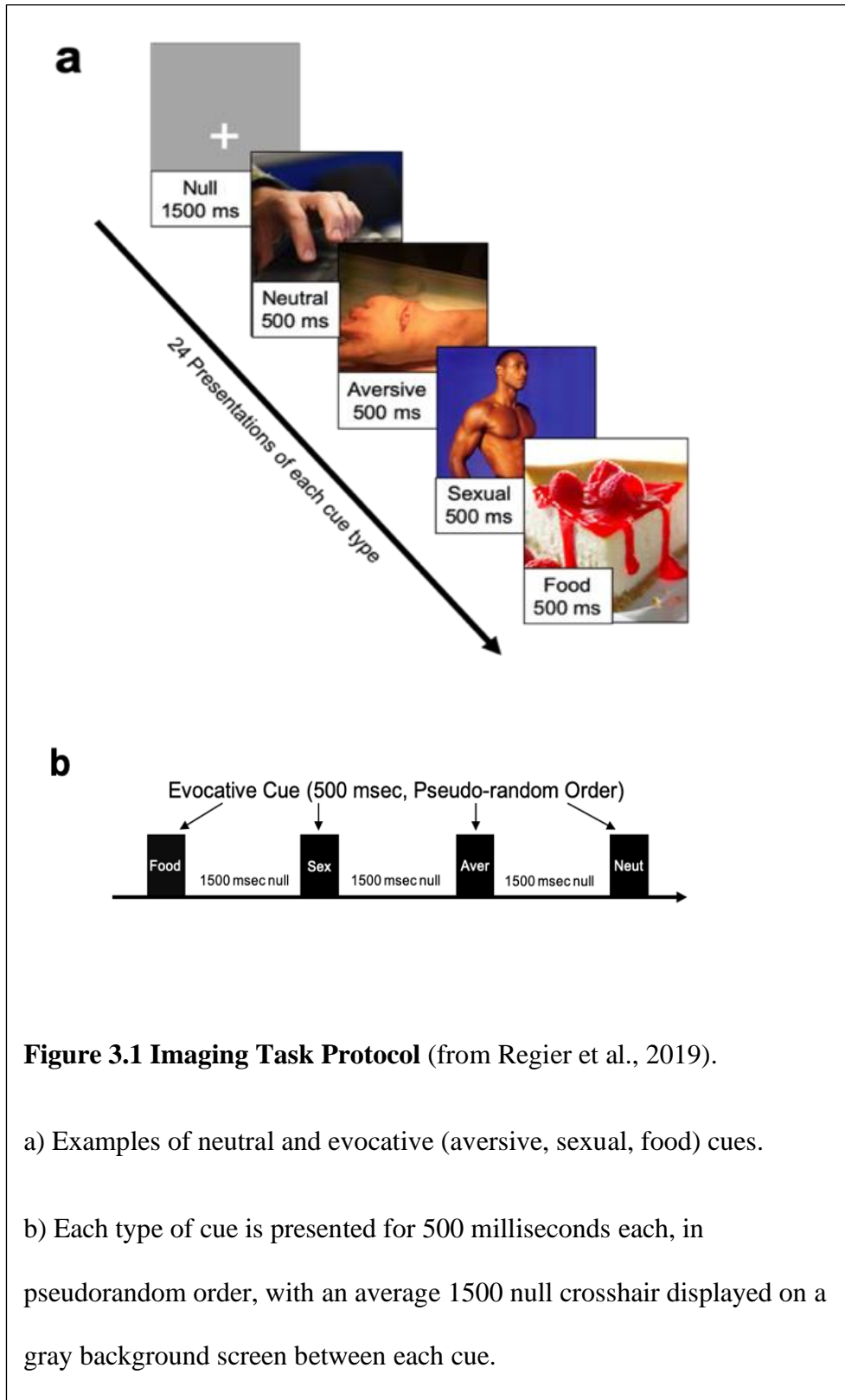
Limitations

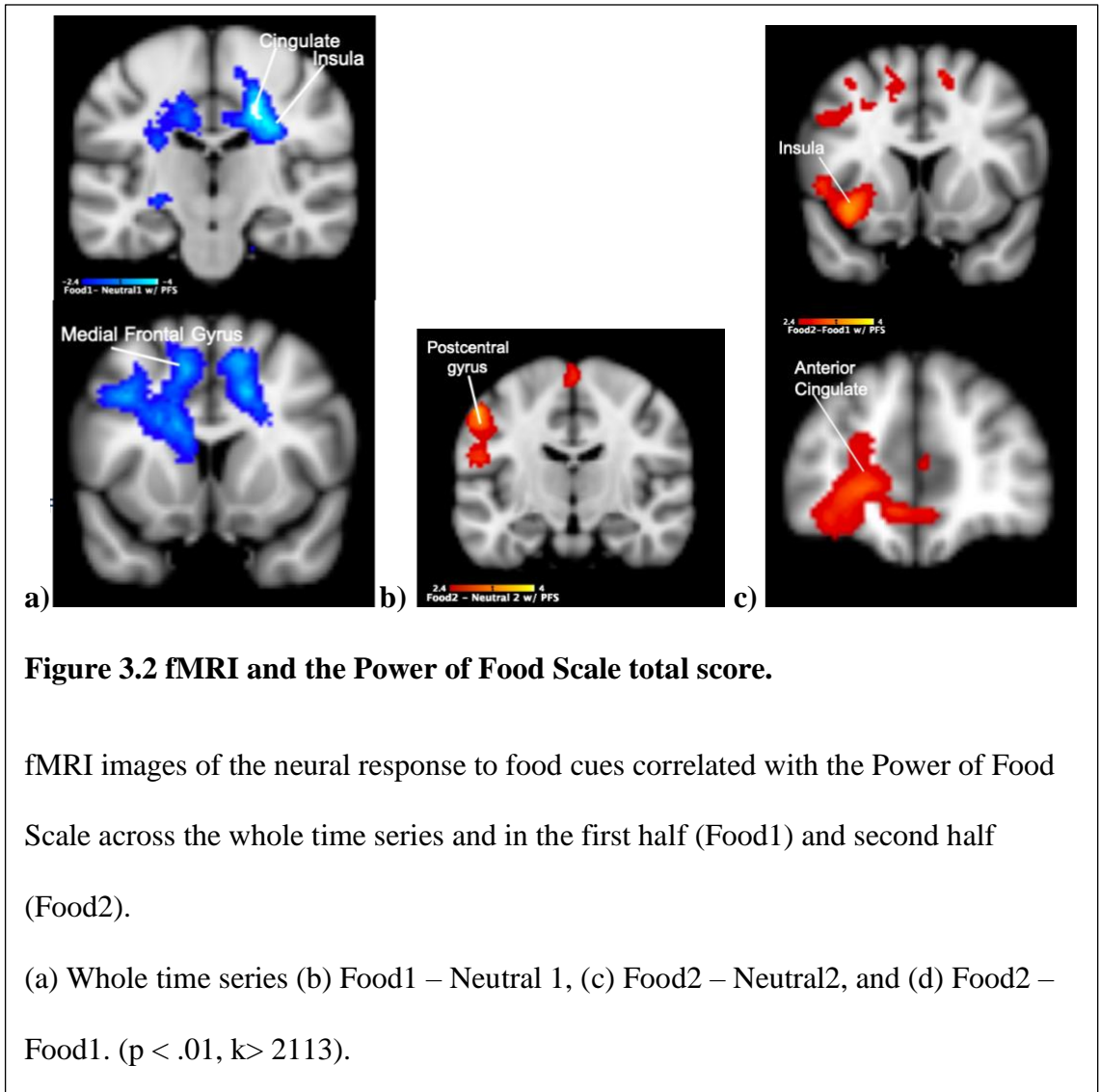
Interpretation of the findings from this study requires careful consideration of the limitations. First, our study included cross sectional data, so we cannot determine

causation or confirm the stability of the relationship over time. Evidence suggests that a person's hunger state does not significantly impact the Power of Food Scale total score (Witt et al., 2014), but it may impact the neural response to food cues (Kennedy & Dimitropoulos, 2014). Second, this was a secondary analysis, so this study was limited by a given set of variables. Additional data regarding disordered eating behaviors or eating disorders may have helped better explain the results. Finally, although experiences of maltreatment may impact brain development, there is evidence that resilience or the ability to adapt and cope can prevent or lessen the poor health outcomes associated with adversity (Kalisch et al., 2017). However, the evaluation of temporal dynamics in a demographically diverse sample is different from previous research and add to the literature related to the neurobiological study of food cues and obesity.

Future Directions

Future studies should quantify the relationship between the neural response to repeated cues and motivation to eat measured by the Power of Food Scale score and eating behavior. Accounting for food insecurity in statistical models may help further explain this relationship and sample sizes should be large enough to achieve adequate power. In addition, characterizing resilience or factors that protect from the deleterious effects of childhood maltreatment may help to explain differences in neural response. It would be of further interest to explore how the different types of abuse and subscales of the Power of Food Scale are related to neural response to food cues.





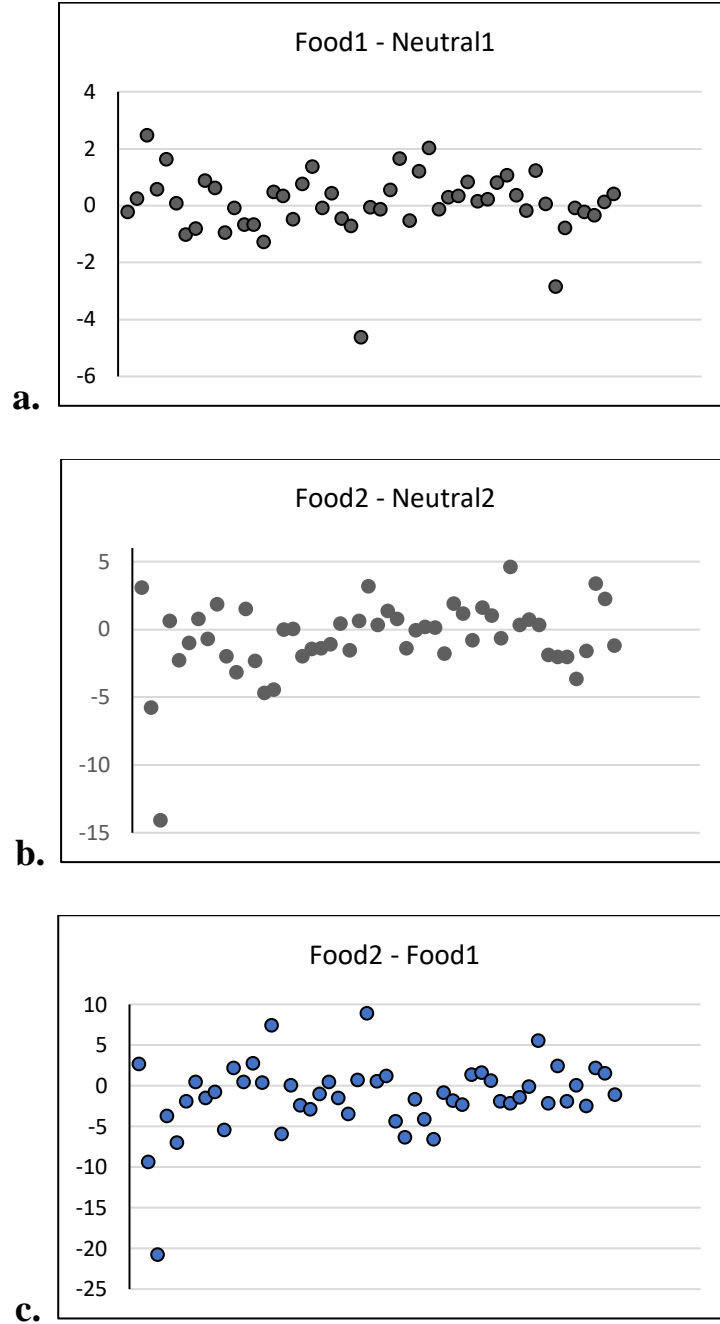


Figure 3.3 Beta values from significant clusters.

Beta values were extracted from significant clusters in the fMRI cue task

(a) Food1 – Neutral1, (b) Food2 – Neutral2, (c) Food2 – Food1.

Table 3.1 Demographics and Health Data (n = 52).

Demographics/Health Data		Percent (%) or Range
Age (years) Mean (SD)	20.9 (2.5)	18 - 24
Race (n)		
<i>White</i>	10	19
<i>Black/African American</i>	31	60
<i>Asian</i>	4	8
<i>Multiracial/Other</i>	7	13
Hispanic (n)	9	17
Student Status (n)		
<i>Yes</i>	20	38
<i>No</i>	32	62
BMI (n=47) Mean (SD)	25.63 (7.5)	17.5 - 66.1
Underweight (n)	2	4% (17.5-18.2)
Normal (n)	28	60% (18.6-24.7)
Overweight (n)	10	21% (26.43- 29.63)
Obese (n)	6	13% (30.07- 39.8)
Severe Obese (n)	1	2% (66.1)
Power of Food Scale Mean (SD)	43 (15)	15 - 75
Food Insecurity Mean (SD)	4.4 (1.8)	3 - 10
CES-D₁ Mean (SD)	17 (11)	4 - 49
CTQ₂ Mean (SD)	39 (16)	25 - 89

¹Center for Epidemiologic Studies Depression Scale (range 0-60). Higher scores indicate presence of more symptomatology. ²Childhood Trauma Questionnaire (range 25- 140). Higher scores indicate more frequent experiences of abuse.

Table 3.2 Bivariate correlations among BMI, depression, child abuse, and food-related variables.

	1	2	3	4	5	6	7
1. BMI	1						
2. Power of Food Scale	0.088	1					
3. Food Insecurity #1	-0.099	0.235	1				
4. Food Insecurity #2	-0.037	.355*	.596**	1			
5. Food Insecurity #3	-0.042	0.196	.578**	.480**	1		
6. CTQ	0.174	0.197	0.265	.384**	0.201	1	
7. CES-D	-0.035	0.253	.352*	.459**	.359**	.502**	1

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 3.3 Significant Clusters and the Power of Food Scale. Significant clusters from observation of food cues compared to neutral with the area identification, correlates of the area, and peak T values.

	Cluster Size	p- Value	Area/Node	x	y	z	Peak T value
Food1 - Neutral1	9938	p < .01	cingulate				
			gyrus	22	-19	37	4.17
			insula	30	-21	29	3.86
			medial frontal gyrus	-12	9	53	3.67
Food2 - Neutral2	3966	p < .01	post central gyrus	-54	-13	47	3.72
Food2 - Food1	3037	p < .01	insula	-34	13	-9	3.26
			anterior cingulate	-20	49	1	2.94

Table 3.4 Model Summary.

Summary of the model exploring the interaction between neural response to food cues and maltreatment using beta values from significant clusters (a) Food1- Neutral, (b) Food2 -Neutral2, (c) Food2 – Food1.

a)

Model Summary Food1- Neutral1									
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.502 ^a	0.252	0.221	13.08037	0.252	8.091	2	48	0.001
2	.516 ^b	0.266	0.219	13.09565	0.014	0.888	1	47	0.351

a. Predictors: (Constant), CTQ, Food1Neut1_wholeCluster

b. Predictors: (Constant), CTQ, Food1Neut1_wholeCluster, MaltxF1N1

b)

Model Summary Food2 - Neutral2									
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.466 ^a	0.217	0.184	13.38413	0.217	6.651	2	48	0.003
2	.467 ^b	0.218	0.168	13.51931	0.001	0.045	1	47	0.833

a. Predictors: (Constant), CTQ, Food2Neut2_wholeCluster

b. Predictors: (Constant), CTQ, Food2Neut2_wholeCluster, MaltxF2N1

c)

Model Summary Food2 - Food1									
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.459 ^a	0.211	0.178	13.43617	0.211	6.414	2	48	0.003
2	.464 ^b	0.215	0.165	13.54350	0.004	0.242	1	47	0.625

a. Predictors: (Constant), CTQ, Food2Food1_wholeCluster

b. Predictors: (Constant), CTQ, Food2Food1_wholeCluster, MaltxF2F1

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Chapter 4

An fMRI Study of Food Cue Reactivity and Impulsivity in Emerging Adult Women

Abstract

Obesity has been associated with deficits in behavioral inhibition that results in increased impulsive behavior. Impulsivity is associated with increased caloric intake, overeating, binge eating, and deficits in the prefrontal cortex of the brain. Evidence shows that this area is also impacted by experiences of childhood maltreatment. However, the exact relationship between these deficits and neural response to food cues is inconsistent. The purpose of this study was to examine the relationship between the neural response to food cues and inhibition and impulsivity in emerging adult women. An exploratory aim examined the moderating effects of experiences of childhood maltreatment. This study used the Behavior in Urban Female Risk Study which explored the relationship between neural response to appetitive and aversive cue and sexual risk behaviors. A secondary analysis was conducted to explore the association between neural response to food cues and the Barratt Impulsivity Scale score and errors of commission on the Affect-Congruent Go/NoGo task. There were no significant correlations between the neural response to food cues and the Barratt Impulsivity Scale score or the Affect-Congruent Go/NoGo task, therefore a moderation analysis was not warranted. Despite these negative findings, future studies utilizing food specific tasks may better explain the relationship between vulnerabilities for obesity and food cue reactivity.

Introduction

Obesity is characterized by increased adiposity and is defined as a body mass index (BMI) greater than 30 kg/m² (World Health Organization, 2018). A problem of epidemic proportions, obesity is prevalent in approximately 40% or 93.3 million adults in the United States (Hales et al., 2017) with notable disparities based on gender and socioeconomic status. Women from lower socioeconomic status have greater rates of obesity than women from higher socioeconomic status (Ogden et al., 2017). These associations are not seen in men. A prevailing hypothesis for the cause of excess weight gain is an imbalance between food consumption and energy exertion. However, given the current obesogenic environment of easily accessible and highly palatable foods and the speed at which the epidemic has progressed, it is believed that overconsumption of food contributes more to this epidemic than lack of energy exertion or genetics (Berthoud, 2007; Mokdad et al., 1999). As a result, current research explores the biological basis for behavior to explain the rapid rise in obesity.

Studies on obesity and weight gain have suggested that deficits in inhibitory control contribute to this epidemic (Appelhans, 2009). Inhibitory control is an executive function that modulates resisting temptation or impulsive behavior and is associated the frontal cortex region of the brain (Dawe & Loxton, 2004; Diamond, 2013; W. Hofmann et al., 2009; Metcalfe & Mischel, 1999). In response to pictures of palatable foods (food cues), individuals previously with obesity who maintained normal weight for > 3 years showed greater activation in the frontal cortex areas associated with inhibition compared to people of normal weight and people currently with obesity. This finding suggests that enhanced inhibitory function may prevent overeating and weight gain (McCaffery et al.,

2009). Also, compared to normal weight individuals, people with obesity demonstrated greater activation in the right middle and right inferior frontal gyri (Garavan et al., 1999), areas associated with integration of reward signals with executive function, which may explain why some individuals have greater difficulty resisting tempting stimuli like food.

Impulsivity is associated with poor planning and rapid action without regard for the possible negative consequences of the actions (Moeller et al., 2001). Behaviorally, this manifests as inattention, lack of perseverance, sensation-seeking, and risk-taking and is associated with excessive food intake, binge eating and obesity (Aoun et al., 2019; Davis & Carter, 2009; Joseph et al., 2011; Nederkoorn, Braet, et al., 2006; Nederkoorn et al., 2007; Nederkoorn, Smulders, et al., 2006; Pearson et al., 2015). Impulsivity also correlates positively with caloric intake (Guerrieri, Nederkoorn, & Jansen, 2007; Guerrieri, Nederkoorn, Stankiewicz, et al., 2007), activation of reward circuitry in the mesolimbic brain region (Beaver et al., 2006), greater attentional bias to food (Bongers et al., 2015) and higher BMI scores (Bénard et al., 2017; Braet et al., 2007; Ryden et al., 2003). Impulsivity scores also correlate negatively with weight loss during obesity treatment (Nederkoorn, Smulders, et al., 2006; Yeo et al., 2020).

Experiences of maltreatment in childhood like physical, sexual, or emotional abuse and neglect, have been associated with increased risk for obesity in adulthood, specifically in women (Danese & Tan, 2014; Hemmingsson et al., 2014). Emerging adulthood is a unique developmental period characterized by instability of housing, finances, and identity, which some believe is the most critical development during this time period (Arnett et al., 2014). In addition to these changes, this period is marked by rapid physiological changes to the areas of the brain associated with executive function

and inhibition of impulsive behavior (Blakemore, 2012). Because of these changes, it is believed the effects of childhood maltreatment become more salient during this time period (Wright et al., 2009) and may contribute to decreased inhibitory function and increased vulnerability to rewarding stimuli (Novick et al., 2018).

The purpose of this study is to examine the relationship between the neural response to food cues using functional magnetic resonance imaging (fMRI) and inhibition as measured by the Affect-Congruent Go/NoGo (GNG) task errors of commission and impulsivity as measured by the self-report questionnaire Barrett Impulsivity Scale (BIS). It is hypothesized that in response to observation of palatable food cues, compared to neutral cues, greater levels of inhibition and impulsivity will be associated with increased activation in the brain, specifically the dorsomedial prefrontal cortex, and decreased activation in the medial frontal gyrus and superior frontal gyrus. An exploratory aim will examine if this relationship is moderated by experiences of maltreatment in childhood, while controlling for depression and a measure of food insecurity, both known to be associated with experiences of childhood maltreatment. Furthermore, food insecurity is not often controlled for in studies of the neurobiological basis of eating.

Methods

Study Design

A secondary data analysis of the Behavior in Urban Female Risk Study was performed. The parent study was designed to examine the neural response to neutral and evocative cues (food, sex, aversive) in emerging adult women with varying degrees of risk for sexually transmitted infections (STIs)/HIV infection. Participants (n= 60) were recruited from a federally supported Title X (serving low income individuals) family planning

clinic and from a nearby university, both of which were located in a large urban area in the United States.

Subjects participated in two sessions, typically scheduled on consecutive days. In the first session, participants completed informed consent, surveys, and demographic health information via pen and paper. For sensitive topics like intimate partner violence and adverse childhood experiences, Audio Computer Assisted Self-Interviewing (ACASI) was utilized for data collection in order to increase the accuracy of self-report data (Morrison-Beedy et al., 2006). In the second session, an fMRI scan was completed while performing selected tasks on a computer. Following completion of the study, participants were compensated 105 dollars. This study was approved by the Institutional Review Board approval prior to initiation.

Sample

A convenience sample of participants were selected for the BUFR 2 study. From the original sample, 52 subjects were included. Inclusion criteria were age 18-24 years, female gender, a BIS score, completion of Affect- Congruent Go/NoGo task, and an fMRI scan that included the cue reactivity task. Subjects (n= 8) were excluded due to incomplete fMRI data.

Survey Measures

Barratt Impulsiveness Scale (BIS; Patton et al., 1995). This 30-item questionnaire measured impulsiveness through items such as “I act on impulse” and “I consider myself always careful”. Participants indicated how frequently each statement applies to them on a 1 to 4-point Likert scale (*never, occasionally, often, almost always*). Possible score totals ranged from 30 to 120, with higher scores indicating greater total levels of impulsiveness. There are six first-order factors (i.e., attentional, cognitive complexity,

cognitive instability, motor, perseverance, self-control) which load onto three higher order factors (i.e., attentional impulsiveness [attention + cognitive instability], motor impulsiveness [motor + perseverance], non-planning impulsiveness [cognitive complexity + self-control]). For the current study, only the three higher order factors and total score were considered. The BIS has acceptable internal consistency as indicated by Cronbach's alphas ranging from .79 to .83 (Patton et al., 1995). The Cronbach's alpha for this sample was 0.70.

Child Maltreatment. The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1997) is a self-report measure that includes 28 items to rate the severity of emotional abuse and neglect, physical abuse and neglect and sexual abuse. Items are rated on a 5-point scale ranging from 1 (never true) to 5 (very often true). The sum of the five items for each subscale ranges from 5 to 25. Moderate to severe abuse is indicated by scores 13-15 for emotional abuse, 10-12 for physical abuse, 8-12 for emotional abuse, 15-17 for emotional neglect, and 10-12 for physical neglect. Cronbach's alpha for the factors ranged from 0.79 to 0.94, indicating high internal consistency. The CTQ also demonstrated good test-retest reliability over a 2- to 6-month interval (intraclass correlation = 0.88) (Bernstein et al., 1997). The Cronbach's alpha for this sample is 0.766.

Depression. The Center for Epidemiological Studies-Depression (CES-D) (Radloff, 1977), is a 20-item measure that asks participants to rate how often over the past week they experienced symptoms associated with depression, such as restless sleep, poor appetite, and feeling lonely. Response options range from 0 to 3 for each item (0 = Rarely or None of the Time, 1 = Some or Little of the Time, 2 = Moderately or Much of the time, 3 = Most or Almost All the Time). Scores range from 0 to 60, with high scores

indicating greater depressive symptoms. It has a reliability of 0.85 across diverse populations (Lewinsohn et al., 1997). The Cronbach's alpha for this sample was 0.84.

Food Insecurity. Three questions assessed food insecurity: 1) Do you ever have to make choices between spending money on food or spending money on other needs? (i.e. medication, utilities, rent, etc.); 2) Do you ever worry whether your food will run out before you have money to buy more?; 3) In the last three months, about how many times have you skipped a meal or cut the size of your meal because you do not have enough money for food? For question 1 and 2 the options are 1 (never), 2 (sometimes), 3 (often). For question 3, response options ranged 1 to 5 (1= 0 skipped meals, 2= 2-5, 3= 6-10, 4= 11-20, 5 >20). Question 1 and 2 have a 97% sensitivity for food insecurity indicated by scores of often or sometimes true compared to never true (Hager et al., 2010). For this analysis we combined the three questions with possible responses ranging from 3 to 11, with binary cutoff of 3 = no food insecurity and a total score > 3 = food insecurity. The Cronbach's alpha for this sample was 0.779.

Demographic and Health Variables. Data on participants age, current education status, race (Caucasian/White, African American/Black, Native American, Asian/Pacific Islander, Other), and ethnicity (Hispanic/Latino) were collected.

Behavioral Task

Affect Congruent Go/NoGo. The Affect-Congruent Go/NoGo task, a measure of inhibition described by Goldman et al. (2015), was administered via a laptop computer programed using "E-Prime" (Psychology Software Tools, Pittsburgh, PA). This task goes beyond simple motor inhibition and is designed using stimuli that reflect real world signals for reward and danger. Go stimuli were pictures from one of three categories: baby animals,

sweets, and flowers; NoGo stimuli were pictures from one of three categories: scorpions, snakes, and spiders. The task consisted of three levels of difficulty: the most difficult condition had 12.5% NoGo's (with 20 NoGo trials and 140 Go trials; 2.7 minutes), the moderate condition had 25% NoGo's (with 20 NoGo trials and 60 Go trials; 1.3 minutes) and the least difficult condition had 33% NoGo's (with 20 NoGo trials and 40 Go trials; 1 minute). Maintaining the same number of NoGo trials (20) allowed for direct comparison of error rates across the three conditions. To optimize the prepotency load, the Go and NoGo trials were randomly presented with the constraint that 1 NoGo trial would occur within each block of 8 trials in the 12.5% condition, within each block of 4 trials for the 25% condition, and within each block of 3 trials for the 33% condition. To reduce carry-over across conditions we repeated the instruction screens before each condition. Each trial began with a 500 msec stimulus presentation followed by a 700 msec presentation of a cross-hair fixation point. The participants could respond at any point during the 1 sec trial. Participants completed a practice session prior to completing the experimental session.

Imaging. Participants completed a blood-oxygen-level dependent (BOLD) functional fMRI scan. The imaging center contains a Siemens 3 Tesla (Trio) research-dedicated magnet, an 8-channel head-coil, an LCD projector for stimulus presentation, air-conducting earphones, and a fiber optic response pad. Mirrors, attached to the head coil, are adjusted so that participants can focus attention on projected stimuli and instructions. Prior to the functional scans, a 3 min localizer scan and a T1-weighted high resolution resting scan (5 min) were acquired. For functional scans: T2*-weighted BOLD images

were obtained with single shot gradient echo planar imaging sequence (field of view = 192 mm, matrix 64x64, TR=2 sec, TE=30ms, flip angle=80).

Data Analysis

Descriptive statistics (mean, standard deviation, median, range, and percentiles) were produced to describe the sample characteristics. All data were closely examined for systemic patterns of missingness. Pearson's correlation coefficients were assessed to determine the relationships among variables of interest. All statistical analyses were performed using SPSS software version 25.0.

FMRI data were preprocessed using a standard Statistical Parametric Mapping 12 (SPM 12; Wellcome Department of Cognitive Neurology, 2014) batch mode scripts modified from ASLtbx. Basic steps, including slice-timing correction, motion correction, temporal filtering, spatial smoothing, and registration to the Montreal Neurological Institute (MNI) standard brain, were processed using a standard SPM12 pipeline. The motion time courses were further removed from fMRI data using simple regression. Spatial smoothing was performed with an 8 mm³ full-width half-maximum Gaussian kernel.

Statistical analyses used a general linear model with a canonical hemodynamic response function with both the first (time) and the second (dispersion) derivatives using SPM 12 and MATLAB (Mathworks, 2019a). Three first-level contrasts [food minus neutral in first and second halves of the task (food1-neut1, food2 -neut2), food second half (f2) minus food first half (f1)] were defined to assess the effects of repeated cue presentation correlated with the Barratt Impulsivity Score and the Affect Congruent Go/NoGo errors of commission. Clusters were considered significant at $p < 0.01$, cluster-corrected with Analysis of Functional Neuroimages (AFNI version 20.0.19) program

3dClustSim (Statistical and Scientific Computing Core, 2019). Parameter estimates were extracted from significant clusters within regions of interest to be used in the moderation analysis. The cue-rater was also correlated with the neural response to the food cues presented in the task.

Data for the go/no task were extracted from E-Prime and data analysis was performed in SPSS software version 25.0 (IBM Corp., NY). Errors of commission were correlated with imaging data using Pearson's correlation coefficient.

To examine the impact of childhood maltreatment on the relationship between neural responses to food cues and impulsivity, the Baron and Kenny (1986) moderation analysis were applied. A multiple linear regression was conducted. The independent variable of the regression was neural response to food cues as beta values extracted from significant nodes and childhood maltreatment. The interaction was created by multiplying the beta values from selected regions and childhood maltreatment together after both have been centered to have a mean of 0. The dependent variable of the regression was impulsivity/inhibition. If the interaction is significant, then moderation is supported. The BIS score and the errors of commission from the Affect- Congruent Go/NoGo task were the dependent variables. Based on correlations, control variables included food insecurity question 2 and CES-D total score.

Results

Demographic characteristics and means and *SDs* of primary study variables are reported in **Table 4.1**. The sample consisted of 52 women with an average age of 20.9 years (*sd*; standard deviation 2.5). Sixty percent (60%) of the sample self-identified as Black or African American. A majority of participants were not students (62% v. 38%).

BMI data was collected for 47 participants and 60% of the sample had a normal BMI. The depression scores as measured by CES-D ranged 4-49 and the impulsivity scores as measured by BIS ranged 43-101. The average score for the total food insecurity questions was 4.4 (s.d. 1.7). Twenty-eight participants endorsed some level of food insecurity (total score >3). Average errors of commission for the three conditions (12%, 25%, 33%) on the Affect-Congruent Go/NoGo task were 6.76 (sd 2.9), 6.12 (sd 3.0), and 5.44 (sd 3.1).

We sought to explore the relationship between the neural response to food cues visualized by fMRI and Affect-Congruent Go/NoGo task, a measure of response inhibition, and the BIS measure of impulsivity. After applying AFNI correction standards using a frontal lobe mask, there were also no significant correlations between inhibition as measured by the Affective Go/NoGo task errors of commission and the neural response to food cues. Further, there were no significant clusters present when analyzing the correlation between the BIS total score of impulsivity and neural response to food cues in the whole time series. No significant clusters were present when analyzing the differences between first and second presentation of food cues compared to neutral and comparing first and second presentation of food cues to each other.

Because there was no significant relationship between the neural response to food cues and the affective Go/NoGo task or the BIS, a moderation analysis is not warranted. However, there were significant correlations between the BIS total score and maltreatment ($p < .01$) and depression ($p < .01$). There were also significant correlations with the maltreatment score and food insecurity ($p < .01$). Correlations of variables of interest are found in **Table 4.2**.

Discussion

In this study, we sought to explore the relationship between the neural response to food cues with a measure of inhibition and impulsivity in a group of emerging adult women. We found no significant relationship between the neural response to food cues and inhibition as measured by the Go/NoGo task or impulsivity measured by the self-reported BIS. There were also no significant correlations between the Affective Go/NoGo Errors of Commission at any level of difficulty (12%, 25%, 33%) and the BIS. There are several possible explanations for why our results may differ from previously reported studies due to differences in the presentation of food cues, measures of inhibition and impulsivity, neurodevelopmental changes and hunger state.

First, we compared palatable food cues like cake and French fries to neutral cues such as a lamp or shoe. Previous studies compared differences in neural response to high and low calorie food cues like pizza and salad (Meule et al., 2014), to bland foods like uncooked rice or potatoes (Beaver et al., 2006), or gustatory cues like a milkshake (Babbs et al., 2013), which likely accounts for our differences in results. We were only interested in how the brain responds to palatable food cues. Furthermore, there is evidence to suggest that the sensory properties of food elicit different responses in the brain (Rolls, 2005), so it is impossible to compare visual and gustatory cues.

Second, there are several ways to measure behavioral inhibition and impulsivity. One previous study utilized the Go/NoGo task and BIS, but did not examine the neural response to food cues, rather examined the neural response to errors of commission during the task (Hsu et al., 2017). Similar to our study, this study did not find a correlation between Go/NoGo errors of commission and BIS. Further, our Go/NoGo

paradigm utilized stimuli such as puppies as “Go” stimuli and “spiders” as “No/Go” while the other studies have utilized food specific stimuli for Go and No/Go (Meule et al., 2014). When using food specific stimuli, they found a positive correlation with errors of commission and high calorie food compared to low calorie food, suggesting more difficulty resisting the pull of more rewarding stimuli. Though our paradigm does use some food cues as go stimuli, they were used in conjunction with other positive affect stimuli. Finally, others have used the Stop-Signal Task (Bartholdy et al., 2016; Watson et al., 2017), which is similar to the Go/NoGo task in that both require behavioral inhibition of a prepotent response to specific stimuli, however, it is hypothesized that the neural mechanism of inhibition is different for these tasks (Raud et al., 2020). Therefore, it would be difficult to interpret these comparisons. Lastly, our study utilized the BIS as a measure of impulsivity, which some consider to be a trait vs. a state that can be induced (Guerrieri, Nederkoorn, Stankiewicz, et al., 2007). Our study only measured the trait form, so it is possible that the neural signatures of these two forms of impulsivity may be different.

Third, age related brain development may also contribute to differences in results. Previous studies have found that hypoactivation in regions associated with inhibition and impulsivity were associated with increased BMI, however compared to our sample (average age of 21), this sample was adolescents females of average age about 16 (Batterink et al., 2010). Another study found that adolescents with greater impulsivity had greater sensitivity to visual food cues, but this study utilized event-related potential recordings instead of fMRI (Hofmann et al., 2015). Given that the brain is still developing through age 26, with particular development during this time focused on the

frontal lobe (Blakemore, 2012), it is possible that age may account for our differences. To further test this assumption, studies should examine longitudinal differences in impulsivity and how that relates to sensitivity to food cues.

Lastly, our study contained no information related to hunger state. Previous studies have found neurofunctional differences in people with obesity compared to normal weight people (for a review, Kennedy & Dimitropoulos, 2014). Some studies have demonstrated that hunger state may impact blood flow to areas like the prefrontal cortex (Del Parigi et al., 2002) and or lead to increased activation the medial frontal gyrus (Smeets et al., 2006), which are regions associated with inhibition and impulsivity, however it has been proposed that regardless of hunger state, individuals with obesity may be more sensitive to palatable food cues. In individuals with obesity, these cues have the ability to override previous conscious decisions to refrain from eating (Tuulari et al., 2015). Moreover, differential brain responses to food cues have been shown in women that were actively dieting or have a history of dieting regardless of weight status (Ely et al., 2014; Feig et al., 2017), so it would be important to include this assessment in future studies.

An exploratory aim of this study was to examine childhood maltreatment as a moderating factor in the relationship between neural response to food cues and inhibition and impulsivity. Because we did not find any correlation in our analysis of the neural response to food cues and the BIS and Go/NoGo, the relationship was not explored. Notably, the BIS score did correlate with our measure of depression and maltreatment, while only the Affective Go/NoGo 12% correlated with inversely with total maltreatment score suggesting that a relationship between inhibition, impulsivity, and maltreatment

does exist. However, the exact relationship is still unclear. Previous studies show an association between childhood maltreatment and greater levels of impulsivity (Oshri et al., 2015; Ouyang et al., 2008), depression (Cohen et al., 2017), increased odds of obesity (Alvarez et al., 2007; Rohde et al., 2008) and food insecurity (Chilton et al., 2017; Chilton et al., 2014; Pryor et al., 2016). Given the associations between these variables, future research exploring neural response to food cues and impulsivity should include measures for food insecurity, depression, and child maltreatment.

Limitations

Our study was not without limitations. Due to the nature of the cue-reactive task, this study may have been impacted by selection bias as it required participants to sit without moving in an enclosed imaging device. Participants with claustrophobia or an inability to sit for long periods of time may have chosen not to participate. Due to the structural limitations of the neuroimaging device, participants were restricted to < 300 pounds, thus eliminating those with higher BMI. There was no information regarding hunger state, so it is possible that this may have impacted neural responding to food cues (Kennedy & Dimitropoulos, 2014). Also, our Go/NoGo task was not specific to food and included a variety of appetitive and aversive stimuli. Although there were food cues included and stimuli were affect-congruent for the go (positive stimuli) and NoGo (negative) conditions. Finally, we did not evaluate reaction time for the Go/NoGo data, but this may help to better explain processing of stimuli and interpretation of which cues (affective/aversive) are more likely to contribute to errors of commission (Meule, 2017). Knowledge of this information may also help with interpretation of the neural response to food cues.

Future Directions

Future studies should evaluate reaction time on the Go/NoGo task and employ a food specific Go/NoGo task. Completion of the Go/NoGo task during fMRI scanning will further enhance our understanding of how the brain is responding during motor inhibitory tasks specific to food. Also, repeated measures of impulsivity may also clarify the stability of impulsivity with regard to food and food cues. Incorporation of multiple behavioral and survey measures of impulsivity may also help to better explain the multidimensional trait of impulsivity. Finally, consideration of environmental and social factors that contribute to impulsivity will help further clarify the relationship between neural responding to food cues and impulsivity.

Conclusions

In a sample of emerging adult women from diverse sociodemographic backgrounds, the neural response to food cues did not correlate with inhibition as measured by the Affective Go/NoGo or impulsivity as measured by the BIS. These results suggest that sensitivity to food cues in this study was not related to inhibition or impulsivity. Because there was no association between food cues and impulsivity, a moderation analysis was not necessary. Despite these negative findings, future studies should consider a food specific Go/NoGo task as this may be a better way to evaluate food specific impulsivity.

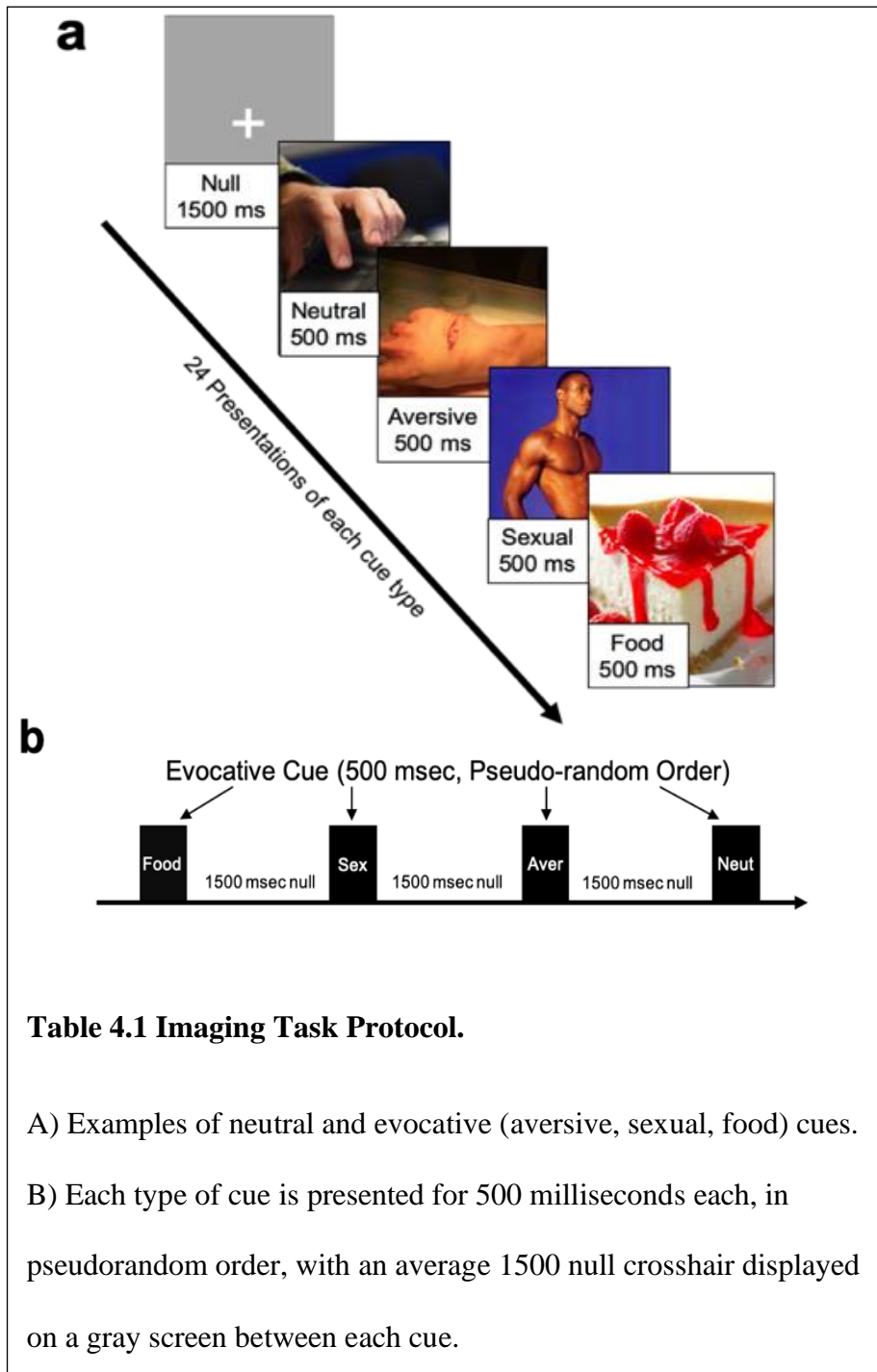


Table 4.1 Demographics and Health Descriptive Data (n=52)

Demographics		Percent (%) or Range
Age (years) Mean (SD)	20.9 (2.5)	18 - 24
Race (n)		
<i>White</i>	10	19
<i>Black/African American</i>	31	60
<i>Asian</i>	4	8
<i>Multiracial/Other</i>	7	13
Hispanic (n)	9	17
Student Status (n)		
<i>Yes</i>	20	38
<i>No</i>	32	62
BMI (n=47) Mean (SD)	25.63 (7.5)	17.5- 66.1
Underweight (n)	2	4% (17.5-18.2)
Normal (n)	28	60% (18.6-24.7)
Overweight (n)	10	21% (26.43- 29.63)
Obese (n)	6	13% (30.07- 39.8)
Severe Obese (n)	1	2% (66.1)
BIS₁ Mean (SD)	60 (11)	43 - 101
GNG₂ Errors of Commission 12% Mean (SD)	6.76 (2.9)	1- 14
GNG₂ Errors of Commission 25% Mean (SD)	6.12 (3.0)	0- 13
GNG₂ Errors of Commission 33% Mean (SD)	5.44 (3.1)	0- 13
CES-D₃ Mean (SD)	17 (11)	4 - 49
CTQ₄ Mean (SD)	39 (16)	25 - 89

¹Barratt Impulsivity Score (range 30-120). Higher scores indicate greater impulsivity.

²Go/NoGo Behavioral Task. ³Center for Epidemiologic Studies Depression Scale (range 0-60).

Higher scores indicate presence of more symptomatology. ⁴Childhood Trauma Questionnaire. (range 25-140) Higher scores indicate more frequent experiences of abuse.

Table 4.2 Bivariate Correlations Among Variables of Interest

	1	2	3	4	5	6	7	8	9	10	11
1. Age	1										
2. BMI	-0.157	1									
3. BIS-10 _f	0.028	0.019	1								
4. GNG ₂ Errors of Commission 12%	0.127	-0.167	0.020	1							
5. GNG ₂ Errors of Commission 25%	-0.083	0.053	0.040	0.639**	1						
6. GNG ₂ Errors of Commission 33%	-0.068	-0.046	-0.060	0.508**	0.662**	1					
7. CTQ ₃	-0.173	0.201	0.430**	-0.277*	-0.044	-0.171	1				
8. Food Insecurity #1	-0.175	-0.092	0.353**	-0.057	-0.014	-0.092	0.279*	1			
9. Food Insecurity #2	-0.030	-0.039	0.197	-0.022	-0.035	0.086	0.372**	0.574**	1		
10. Food Insecurity #3	-0.292*	0.004	0.272*	-0.130	-0.011	-0.005	0.251	0.586**	0.431**	1	
11. CES-D ₄	0.015	0.017	0.467**	-0.261	-0.085	-0.094	0.534**	0.363**	0.432**	.416**	1

¹Barratt Impulsivity Score. ²Go/NoGo Behavioral Task. ³Childhood Trauma Questionnaire. ⁴Center for Epidemiologic Studies Depression Scale

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

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Chapter 5

Discussion

Obesity, classified as a body mass index (BMI) of 30 kg/m² or greater, is a prevalent public health issue that results in 2.8 million premature deaths worldwide each year (World Health Organization, 2018). There are considerable disparities among racial/ethnic minorities and among socioeconomic disadvantaged women who are more likely to suffer from obesity (Ogden et al., 2017). Furthermore, emerging adult women (aged 18-29) are at even more risk, as the rates of obesity are growing faster in this group than any other age group. Due to the variability in treatment efficacy, studies of obesity and eating behaviors have shifted to explore the neurobiological implications in the development and the maintenance of obesity. However, given the association between childhood maltreatment and obesity in emerging adults (Nagata et al., 2018) and the association between childhood maltreatment and altered brain development, studies must consider the impact of maltreatment in neurobiological studies of obesity.

The **overall objectives** of this dissertation research were to explore disordered eating behaviors and its relationship with childhood maltreatment, examine the association between neural response to food cues and vulnerabilities associated with obesity (hedonic hunger, impulsivity and inhibition) and examine how experiences of childhood maltreatment inform the relationship between neural response to food cues and vulnerabilities for obesity.

Chapter 2 of this proposal served as the groundwork for the research aims and hypotheses by providing an increased understanding of the relationship between childhood maltreatment and disordered eating behaviors. Appreciative of deficits in the

neurobiological implications in the development and the maintenance of obesity, **Chapter 3** and **Chapter 4** evaluated data collected from the Brain and Urban Female Risk Study (BUFR 2) which explored how the neural response to food cues relates to “bottom up,” reward driven processes (mesolimbic) and “top down,” inhibitory related processes (mesocortical)- in emerging adult women. Specifically, we measured the correlation between the neural response to food cues and hedonic hunger in **Chapter 3** and impulsivity and inhibition in **Chapter 4**.

The motivation for this work stems from my practice as a registered nurse, experience as a research nurse, and my general interest in motivated behaviors and the brain. As a nurse in a variety of settings, I witnessed the impact of adverse childhood experiences on the physical and mental health of my patients, particularly in people from disadvantaged backgrounds. However, it became clear to me that there were other factors outside of the individual that influenced behavior. It became clear to me that in order to truly understand how the brain regulates behavior, we must understand how the environment and previous experiences shape behavior over time. While there is a large body of literature exploring associations between experiences of childhood maltreatment in emerging adult women with obesity, disordered eating, and eating disorders (Danese & Tan, 2014; Rayworth et al., 2004), few studies explore this relationship how socioeconomic and neighborhood disadvantage, as well as racism may impact neural studies of eating behavior (Mitchison & Hay, 2014). This is of particular concern in studies of brain and behavior because there is ample evidence suggesting that poverty and socioeconomic disadvantage are associated with altered brain development (Briere & Jordan, 2009; Nagata et al., 2018, 2019).

This dissertation research contributes to the existing knowledge that neural vulnerabilities may contribute to the development of disordered eating, hedonic hunger, and obesity. Specifically, there may be underlying differences in the temporal dynamics of neural response to environmental food cues that makes people more vulnerable to overeating and/or binge eating behavior, although this work did not examine that. However, careful consideration of early childhood adversity and social and economic disadvantage is crucial to the advancement of our understanding of the brain's implication in behavior. Importantly, the work brings attention to the challenges of utilizing a neurobiological marker of behavior alone to explain behavior. Continued investigation of the neurobiological contribution to disease will be valuable for future research and is vital to improve the lives of people suffering with obesity.

Major Findings from Chapter 2

There is some evidence supporting the association between disordered eating symptoms and experiences of maltreatment in childhood but the strength and mechanism of that association is unclear. This integrative review of 13 peer-reviewed publications examined the associations between disordered eating symptoms or behaviors, and the role of childhood maltreatment among emerging adult women. The results of this analysis suggest a relationship between disordered eating symptoms and child maltreatment among young adult women aged 18-29. Further exploration of the mechanisms by which disordered eating symptoms can occur in relation to experiences of childhood maltreatment is warranted.

By exploring the relationship between disordered eating behaviors in emerging adult women who have experiences of childhood maltreatment, it was determined that

disordered eating behaviors are prevalent in women with experiences of childhood maltreatment, with particular support for women attending colleges and universities. The behavior most often reported was binge eating behavior. However, the mechanism by which this relationship occurs is still unclear. Future work should focus on examining the neurobiology associated with these behaviors. The results of this integrative review were vital for elucidating the insufficiencies in obesity research that are addressed in **Chapters 3 and 4**.

Major Findings from Chapter 3

Obesity is a prevalent public health issue and current research seeks to identify modifiable risk factors to enhance treatment and prevention. Although some people are able to resist overconsumption of highly palatable, nutrient poor foods (hedonic hunger) others are not. This repeated overconsumption of food can lead to weight gain and obesity. Therefore, it was hypothesized that increased neural response to food cues would be associated with increased hedonic hunger. Specifically, this secondary data analysis examined the association of hedonic hunger, as measured by the Power of Food Scale, with the neural response to food cues measured by fMRI in emerging adult women. A secondary aim was to examine the correlation between the neural response to food cues and the “Food Available” subscale of the Power of Food Scale. This subscale is most associated with motivation for food (Lowe et al., 2009) and most strongly correlated with weight status and weight gain (Lipsky et al., 2016). An additional exploratory aim of this study examines how the relationship between neural response to food cues and the Power of Food Scale may be modulated by experiences of childhood maltreatment.

There were several notable findings. First, we found an inverse relationship between the neural response to novel food cues and Power of Food Scale scores in the cingulate gyrus, medial frontal gyrus, and insula, areas associated with executive control, cognitive evaluation, and interoceptive awareness. Second, we found sustained excitability in the insula and anterior cingulate in when comparing repeated cue presentation to novel cue presentation, suggesting a lack of habituation in these areas. Taken together, these results suggest an association between neural sensitivity to repeated food cues and higher Power of Food Scale scores. There is some degree of food insecurity in this sample, which is known to be associated with obesity and cognitive deficits independently, but it is unclear how this may have altered the results. There was no significant difference in the Power of Food Scale scores for participants with and without food insecurity. There was no support for childhood maltreatment as a moderator of the relationship between neural response to food cues and Power of Food Scale scores.

Major Findings from Chapter 4

There is a paucity of research that explores the neural correlates of obesity with deficits in behavioral inhibition, increased impulsivity, and experiences of childhood maltreatment and the results are inconsistent. The purpose of this secondary data analysis was to examine the relationship between the neural response to food cues and inhibition and impulsivity in emerging adult women. An exploratory aim examined the moderating effects of experiences of childhood maltreatment.

The results of this investigation showed no relationship between the neural response to food cues and impulsivity or inhibition. Although previous studies have found decreased neural response in the areas associated with executive control and inhibition

were associated with obesity and increased craving for food, we did not find these same differences. There was also no indication of a childhood maltreatment as a moderator.

Contributions to the State of the Science

All together, these results present a complex relationship between the brain and risk factors associated with obesity. We found that there was a relationship between the neural response to food cues and hedonic hunger such that people with greater hedonic hunger demonstrated decreased neural response to food cues after first presentation in areas of the brain associated with executive control and interoceptive awareness. These results suggest novel food cues elicit less recruitment in areas of the brain associated with inhibition and internal bodily awareness in people with greater hedonic hunger. This type of response has been found in people with obesity and suggests that of the brain to novel food cues in people with greater hedonic hunger. We also found sustained or increased neural response after repeated presentation in areas of the brain associated with interoceptive awareness and hunger state (Brooks et al., 2013; Stoeckel et al., 2008), suggesting that there may be neural differences in the response to repeated food cues that is associated with greater hedonic hunger. This finding is a new finding in the literature and may contribute to vulnerability to overeating and development of obesity, however, more studies are needed to better understand this relationship. Furthermore, our results did not find any indication that either relationship was moderated by childhood maltreatment despite the findings in previous literature that there may be an association. We did not find an association with the neural response to food cues and impulsivity or inhibition despite other findings in the literature (reference). Because of this result, a moderation is not supported.

The results of this dissertation research in particular highlight the importance of considering repeated exposure to environmental food cues and how that may contribute to increased risk for obesity. In people with neurobiological vulnerability in reward regions of the brain, repeated exposure to food cues may increase the risk of hedonic eating and the development of obesity.

Implications

The findings of this dissertation emphasize the complexity of studying the brain in isolation without understanding the current environmental and social impact on health and behavior, as well as the life course experiences that protect or promote obesity and disordered eating behaviors and symptoms. Clinicians should be aware of the risk for hedonic-driven eating and neural vulnerabilities to reward that contribute to obesity, but should be careful not to overemphasize this relationship, as behavior cannot be simply explained by brain reactivity. Furthermore, screening by nurses and other healthcare providers for disordered eating, especially in economically disadvantaged women with experiences of adversity like maltreatment, should happen early and often to identify patterns that may predict maladaptive eating behaviors. If providers can identify behaviors before reaching clinical threshold, they can introduce interventions to prevent escalation and development of negative health consequences.

The environmental contributions identified in this dissertation are modifiable with systemic and policy change. Food insecurity is known to be associated with lower socioeconomic status, depression, and obesity (Bruening et al., 2017; Townsend et al., 2001). Interestingly, studies have found that even in poor families, mental and physical well-being of the mother contributes to better food security, less family violence, and less

substance use compared to poor women with poor mental and physical health (Gundersen & Ziliak, 2014). In addition to more resources for and better access to food, policy makers should continue to push for more equitable access to healthcare resources to support physical and mental well-being of emerging adult women as this may aid in the prevention of obesity.

Future Directions

The results from this dissertation serve as the foundation for future work in the field of obesity, eating behavior, and childhood adversity. A unique aspect of this study was the exploration of the temporal dynamics of the neural response to food cues and the relationship with hedonic hunger. Previous studies have explored repeated cue presentation (Epstein et al., 2009), but did not use visual food cues nor examine the neural response to cues. This is an important area for future research given the abundance of cues in obesogenic environment.

Another important area for future endeavors is data collection in real-time to better understand the relationship between neural response to food cues and behavior. The Functional Near Infrared Spectroscopy (fNIRS) devices are portable headbands that allow for collection of neurological data outside of the laboratory setting. Utilization of these devices may help better capture how the brain responds to food cues in more realistic settings and test why behavioral interventions may or may not work. This type of data will provide enhanced understanding of individual risk and provide more precise treatment options.

Furthermore, social determinants of health in neurobiological studies of obesity and risk factors are important factors that are often not considered. Figueroa et al. (2020)

proposed that utilization of a Polysocial Risk Score to characterize similar social factors that predict risk for diseases like obesity. Utilizing a score like this will to help better classify the impact of the environmental and social factors that contribute to obesity. Although the development of this score is still in the novel stages utilization of a score like this is particularly important in the study of obesity given the disparities that are so evident.

Finally, it is important to consider resiliency or the factors that protect against the negative health effects of adversity. Experiences of maltreatment in childhood are associated with poor health outcomes, but poor health outcomes do not occur in everyone. Because of the association of maltreatment with disadvantage (socioeconomic and racism) it is even more imperative that we understand how to promote resilience, especially in children, adolescents, and emerging adults who are still experiencing brain development.

Conclusions

In conclusion, this dissertation research contributes to the current understanding of maltreatment and the neural implications of obesity and disordered eating. A literature review and secondary analysis of the BUFR 2 study of emerging adult women revealed an association between experiences of childhood maltreatment and disordered eating behaviors, and an association between the neural response to food cues and hedonic hunger in areas associated with interoceptive sensory awareness and executive control. We found no association between neural response to food cues and measures of impulsivity. Notably, this work highlights the complexity of the study of the long-term

impact of maltreatment on neurobiology, eating behavior, and obesity and provides many avenues for future research.

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Appendix A

Summary of Key Scales for Proposed Study			
Scale/Description	# items / response category/ scoring psychometrics/samples	Reference	Notes
<i>The Power of Food Scale</i> Measure of hedonic eating, eating in the absence of physiological need.	15 items 5 point Likert agreement response choices (1-5) with response options ranging from Never True to Very Often True Mean or sum of scores used for 3 domains and for aggregate scale alpha .81- .91 Capelleri: adults: obese and normal weight, large samples, no ethnic or age breakdown provided Lowe: College students in England and US (Phila.)	Capelleri, JC et. Al (2009). Evaluating the Power of Food Scale in obese subjects and a general sample of individuals: development and measurement properties. <i>International J of Obesity</i> , 33, 913-922 Lowe, MR et al. (2009) The Power of Food Scale. A measure of the psychological influence of the food environment. <i>Appetite</i> , 53, 114-118	3 factors: Food available Food present Food tasted Score 5-75 Distinguish 3 factors and state what they mean, how many questions for each
<i>Barratt Impulsiveness Scale 11 (BIS-11)</i> Assesses personality/behavioral construct of impulsivity	30 items 4 point – frequency response choice, rarely/never, occasionally, often, almost always/always Higher score=greater impulsivity Alpha (total score)= 0.83 from sample of adults (N= 1577; m 393, f 1184)	Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S. L., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt Impulsiveness Scale: An update and review. <i>Personality and Individual Differences</i> , 47(5), 385-395. doi:10.1016/j.paid.2009.04.008	six first-order factors: attention motor self-control cognitive complexity perseverance cognitive instability
<i>Childhood Trauma Questionnaire</i>	28 items 5 subscales	Bernstein et al. 2003. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. <i>Child Abuse & Neglect</i> 27, 169–190	Reverse Score (e.g., never true = 5...) item # 2, 5, 7, 13, 19, 26, 28

Measures 5 categories of childhood trauma experience, including emotional, physical, and sexual abuse as well as emotional and physical neglect	3 validity questions Stem: "As I was growing up... Subscales for physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect. 5-point, Likert scale (1-5) with response options ranging from Never True to Very Often True	alpha reliability for subscales .81 or better except physical neglect scale which ranged .68 - .78 depending on the cohort	Indicate what the higher score indicates
<i>Center for Epidemiologic Studies Depression Scale (CES-D)</i> Measure of depression symptomatology	20 items Never, rarely, sometimes, most/all of the time 0-3 points for each question Alpha >.85	Lewinsohn, P.M., Seeley, J.R., Roberts, R.E., & Allen, N.B. (1997). Center for Epidemiological Studies-Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. <i>Psychology and Aging</i> , 12, 277- 287. Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. <i>Applied Psychological Measurements</i> , 1, 385-401.	Four items need to be reversed scored: # 4, 8, 12, 16 Possible range of scores is zero to 60, with the higher scores indicating the presence of more symptomatology

Appendix B

Summary of Behavioral Tasks			
Task/Description	Method of Evaluation	Reference	Notes
Affect-Congruent Go/NoGo Task to assess inhibitory control. Difficulty inhibiting is reflected in errors of commission. This brain response may be a good biomarker for impulsive behaviors, predicting poor inhibition of “in the moment” responses to cues.	Keyboad letter assigned to “go” and “NoGo.” “Go” pictures are positive (puppies), while the NoGo pictures represent natural danger (scorpions, spiders).	Braver TS, Barch DM, Gray JR, Molfese DL, Snyder A. (2001). Anterior Cingulate Cortex and Response Conflict: Effects of Frequency, Inhibition and Errors. <i>Cereb. Cortex.</i> , 11(9):825-836. Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. <i>Science</i> , 280(5364), 747-749. Forman SD, Dougherty GG, Casey BJ, et al. (2004). Opiate addicts lack error-dependent activation of rostral anterior cingulate. <i>Biol Psychiatry</i> , 55(5), 531-537. Kaufman JN, Ross TJ, Stein EA, Garavan H. (2003). Cingulate hypoactivity in cocaine users during a GO-NOGO task as revealed by event-related functional magnetic resonance imaging. <i>J Neurosci</i> , 23(21), 7839-7843. Goldman et al., 2015)	Future interventions could target ways to modify inhibitory abilities; greater ability to identify risk situations and take precautions to prevent behaviors leading to abnormal eating patterns.
Brief Cue Task to assess reward sensitivity in the brain Passive observation of cues during fMRI	500 millisecond presentation of cues (neutral, food, sex, aversive)	Childress AR, Ehman RN, Wang Z, et al. (2008). Prelude to passion: limbic activation by “unseen” drug and sexual cues. <i>PLoS ONE</i> , 3(1), e1506.	Heightened brain (amygdala, v. striatum / pallidum) and behavioral (affective priming) response to food reward cues. People with decreased sensitivity to food cues may be able to resist the pull of immediate food/hedonic Reward. In an environment of highly palatable food, this restraint enables more time for restraint.

Appendix C

Operational Definitions of Variables				
	Variable	Definition	Measured by	Units range frequency
	Impulsivity	Actions without foresight, poorly conceived.	BIS-11	Score, 30-120, higher score= greater impulsivity
	Inhibition	Ability to stop or prevent response to “NoGo” stimulus	Go-NoGo	# of errors of commission
	Childhood maltreatment	any act of commission or omission by a parent or other caregiver that results in harm, potential harm, or threat of harm to a child. Harm does not need to be intended	Childhood Trauma Questionnaire	Score, 5-25 for each subscale Emotional abuse, Physical abuse, Sexual abuse, Physical neglect, Emotional neglect
	Hedonic Hunger	Hunger in the absence of physiologic need	Power of Food Scale	Score, 15-75, greater score= greater hedonic hunger
	Function brain activity	Measurement of brain activity by detecting changes associated with blood flow. This technique relies on the fact that cerebral blood flow and neuronal activation are coupled. When an area of the brain is in use, blood flow to that region also increases.	Blood-oxygen-level-dependent (BOLD) fMRI	voxels
Covariate	Depression	Mood disorder that affects a person’s sense of well-being	CES-D	Score, 0-60, higher score= greater symptomology
Covariate	BMI	Weight (kg)/Height (m) ²	scale	>18 underweight 18-24 normal weight 25-29 overweight >30 obese
Covariate	Food insecurity	Lack of consistent access to a sufficient quantity of nutritious food.	3 questions that address financial food insecurity.	Score, 3-11, 3= no financial food insecurity, >3 -11 = increasing financial food insecurity
				Data type
				Continuous
				Continuous
				Continuous/ Dichotomous
				Continuous
				Continuous
				Continuous/ Dichotomous
				Continuous/ Dichotomous